Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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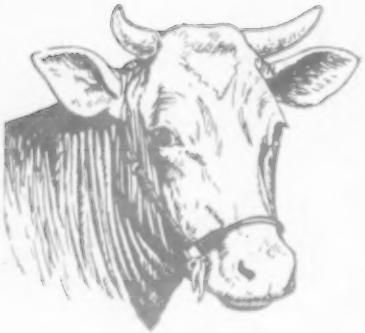

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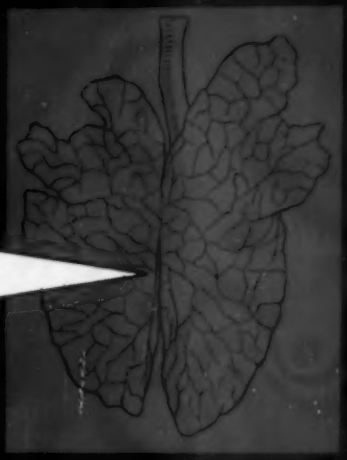

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Correspondence

September 20, 1957

To the Editor:

In the September 15 JOURNAL there are pictures of a radiograph on advertising pages 44 and 45. The "comment" mentions its excellence and encourages others to duplicate it. That is very commendable.

But in figure 2 where the parts are identified, there is the letter "e" for liver and yet on the radiograph there is no letter "e." I think the lower letter "b" on the picture should have been an "e."

It is a trivial thing, but if it is to encourage good technique for others, the example should be beyond criticism.

Very truly yours,
s/Fred J. Wigderson, V.M.D.,
Hackensack, N. J.

[Sorry, the lower *b* should have been an *e*.—ED.]

Hens without roosts, at the Arkansas Experiment Station, laid as many eggs, gained as much, had no higher death losses, and ate no more feed than hens on roosts.—*Farm J. (Sept., 1957): 68A.*

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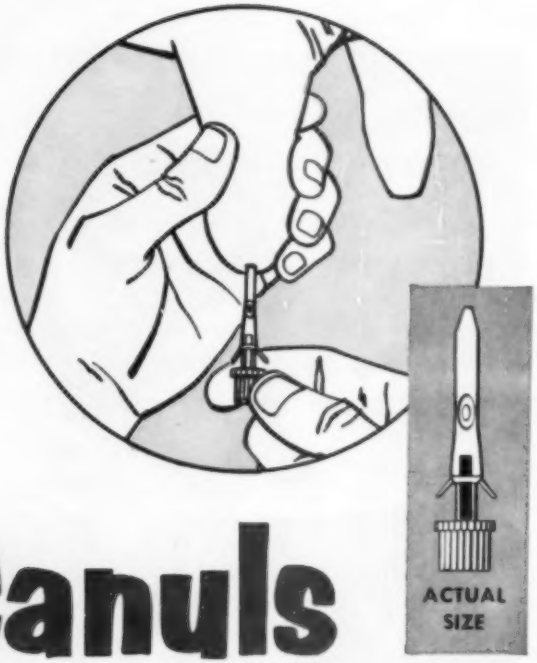

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Public Relations and Professional Liability

Edwin J. Holman, Executive Secretary, Judicial Council, American Medical Association

Presented at the AVMA Public Relations Conference, Cleveland, August 18, 1957

CHAIRMAN KINGMAN: Our luncheon speaker will talk on the subject of "Public Relations and Professional Liability." Last year, at San Antonio, we thoroughly enjoyed and got a great deal from Leo Brown's presentation on public relations.

We are fortunate in again being able to call upon the American Medical Association for a speaker for this year's luncheon. Mr. Edwin J. Holman is executive secretary of the Judicial Council of the American Medical Association. Mr. Holman was born and raised in Leavenworth, Kansas; graduated from the University of Kansas with a degree of LL.B.; practiced law in Leavenworth before coming to the A.M.A. about ten years ago. Mr. Holman!

MR. EDWIN J. HOLMAN: Mr. Chairman, Ladies and Gentlemen: I have a colleague who delights in introducing me by saying, "Ed comes from Leavenworth, Kansas, where he served three terms—in the Kansas Legislature."

As a member of the legal profession, employed by the medical profession, I am extremely happy to appear before the members of your distinguished profession. As a lawyer who has just a little more than a mere acquaintance with the administrative problems of medicine, I feel that you are to be envied. The star of medicine and the star of law has waxed and waned, but veterinary medicine has maintained a consistently high public respect and regard.

My discussion of what relation there may be, if any, between public relations and professional liability I trust will complement the fine remarks made by Dr. Booth this morning. For to say that there is no relation between the two is to shut the door without investigation, and to say there is some relationship may be to advance a postulate rather difficult to prove.

The American Medical Association, as one of a number of professional groups, conducted two surveys within the immediate past few years on public relations and on professional liability.

In the first instance, the association wanted to ascertain, if possible, what people thought of their own doctor, of most doctors, and of the American Medical Association itself as it represents organized medicine.

Also, the association, through its law department, has completed the first stage of a two-stage survey of professional liability, which is a euphonious term for malpractice.

A great many statements have been made, without foundation, concerning this problem of malpractice, and it has become necessary to find out as nearly as possible whether these statements are founded on fact.

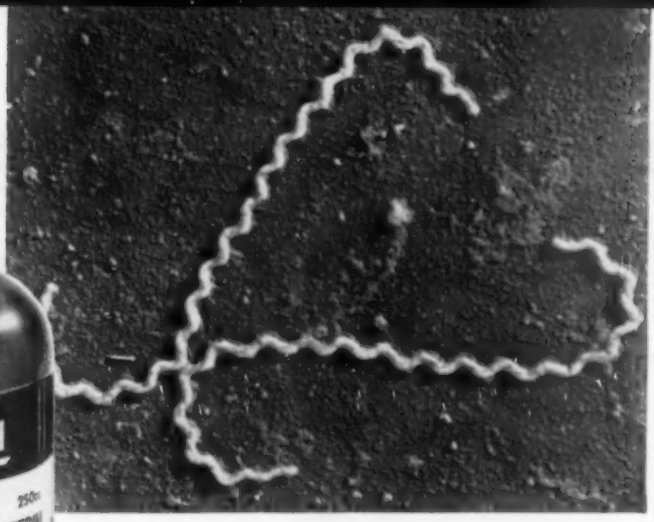
Let me briefly give you a few of the findings from these two surveys and then let us speculate a little concerning the relation between the two. People almost universally like and respect their personal or their family—"my own"—physician. Physicians, as individuals, were given a vote of confidence by the public in this initial survey.

We asked, "Do you like your own doctor as a person?" Ninety-six per cent gave an unequivocal "yes" reply.

Now, a number of questions were asked to determine people's attitude about physicians. The same set of questions was asked twice, first in regard to "my doctor" and then in regard to "most doctors."

The public gave high ratings to family doctors and all doctors on intelligence and capability. And, while people gave "my doctor" a similarly high rating on personal interest, frankness, and amount of time spent with patients, their attitude on these three points toward most doctors was less favorable and showed considerable discrepancy.

For example, 41 per cent of the public say that most doctors keep patients with appointments waiting longer than necessary; 51 per cent say most doctors are hard to reach for emergency calls. And 46 per cent say that most doctors are not frank enough with patients. Sixty per cent of the



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public say most doctors don't give the patient as much time as the patient would like. And yet, only 18 per cent say "This is true of my doctor." About 31 per cent of the public claims that most doctors are too quick to recommend an operation, while only 5 per cent say "This is true of my doctor."

In conducting this survey, certain other questions were also put to doctors themselves. It was found that the public and the doctors agreed rather closely in their appraisal of doctors on intelligence, capability, and willingness to accept medical advance. But, when it comes to dedication to mankind, over-readiness to recommend surgery, belief in one's own infallibility, or lack of personal interest in a patient, doctors' views about doctors represent a position midway between what the public thinks of "my doctor" and "most doctors." In some instances, doctors are much more critical of doctors than the public is. They are inclined to say that physicians do not give patients enough time. Doctors also seem to feel that physicians are not frank enough in discussing the patient's illness; that doctors think they are better than most people, and that most doctors try to cover the mistakes of other medical men.

The American Medical Association, the official voice of the medical profession, has for a number of years occupied a somewhat controversial position on the American scene. It is as often hailed as it is harpooned for its programs and its policies. Any organization which receives so much attention in the public press, both favorable and unfavorable, runs the risk of being stereotyped by the public. Once the imagery of being stereotyped is created, then all of its actions are viewed in the frame of reference of that preconceived image.

But knowledge of the A.M.A. is not as widespread as might be expected. Only half the people say they know anything about it. In answer to an introductory question, 48 per cent of the public say that they have seen or heard or read something about the A.M.A., but only a fifth of those who know anything about the A.M.A. recall anything specific about it. An even lower proportion of those in lower income groups and those with less education say they know anything at all about the A.M.A.

What about our survey on professional liability? Of course, I can hit only some of the high spots. First of all, let me say that professional liability relates to alleged negligence.

When damage directly results to one from the negligent act of another who owes a duty to that one, then the injured person has a cause of action and may resort to the courts in the hope of establishing his claim and obtaining a verdict.

In medicine, generally, this applies to you. The concept is expressed thus: A doctor must possess and exercise that degree of skill and care possessed and exercised by other doctors of his school in like practice, in the same or a similar community.

In the law common to English-speaking people, the first malpractice suit was reported in England in 1374. It is not a new problem. The first reported malpractice case in the United States arose in Connecticut in 1794.

In our questionnaire sent to, and answered by, state medical society executives, we found it was their opinion that the cause of professional liability stemmed from poor results or failure in the following principal fields: surgery; orthopedics, which includes orthopedic surgery; x-ray; diagnosis and therapy, diagnosis generally; general medicine; obstetrics and gynecology. Other areas of medicine followed in a lesser degree.

According to our survey of physicians, 29.7 per cent were of the opinion that there had been an increase in the incidence of professional liability claims in the past five years; 39.7 per cent felt that the incidence of claims had not increased. The remainder thought that claims had decreased or held no opinions.

However, in California, Louisiana, New York, Rhode Island, Utah, and the District of Columbia, there was a clear-cut expression of opinion that professional liability claims had increased in frequency in the past five years.

Our study included an analysis of reported court decisions and it reflected—plus a survey of physicians who stated that a claim had been brought against them—that approximately 50 per cent of claims and suits could not be sustained legally. There were, however, a considerable number of instances reported in which a claim was brought against a qualified physician which involved either actual negligence or a substantial basis on which a patient could reasonably believe he suffered from the negligence of a physician.

In a few instances, it appeared that claims were either fraudulent or so wholly lacking in foundation as to compel the in-

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ference that the patient was acting in bad faith.

Many physicians consider the problem of professional liability as a matter of academic interest. The fact is that professional liability claims are not limited to a small group of malpractice-prone doctors. Among the doctors who indicated that they had experienced claims, 86.5 per cent incurred only one claim in their entire professional practice; 10.5 per cent of the physicians who reported claims had had two claims in their professional career; 1.9 per cent had three claims; and 1.1 per cent had four claims.

Our figures indicate that professional liability is a problem of the many, not the few. In a number of cases which were resolved in favor of physicians because of technical legal problems, it is possible that the verdict would have been against them if the case had been decided on medical merits.

On the other hand, there was a significant number of cases involving the doctor and *res ipsa loquitur*, wherein the courts assumed negligence solely because there was no medical explanation of an unsatisfactory result.

One of the more interesting questions and answers in our survey was the effect of professional liability claims on the physician's reputation. I am not at all sure that physicians will want to accept wholeheartedly the answer we found. A substantial majority of medical society representatives reported that, in their opinion, professional liability claims have little or no effect on reputation or on the practice of the physician involved. A few medical society spokesmen explained that, in smaller communities, the effect of such claims and suits may be more pronounced than in larger communities, particularly if there is a bit of newspaper coverage of the case or the claim.

One of the most disconcerting data reflected by our survey is that the precipitating cause of most claims is the unthinking or unfounded, certainly, nonmalicious criticism of one doctor by another. It is indeed an unfortunate situation, and one that I call particularly to your attention as something to guard against.

Surveys and statistics are wonderful things. We need them; we couldn't do without them; but they cannot be swallowed whole, nor can they be accepted without careful interpretation. Even though one of our surveys indicated that people liked their own doctors and think more highly of "my doctor" than "most doctors," it is none the less true that it is "my doctor" who is the defendant in a malpractice action.

While our malpractice study is not complete, we still have phase 2 to conduct. It is reasonable to state at this time that the

The Preconvention Conference Luncheon, Held August 18, 1957, at the Cleveland Hotel, Where Dr. Edwin J. Holman of the A.M.A. Judicial Council, Spoke on "Public Relations and Professional Liability"

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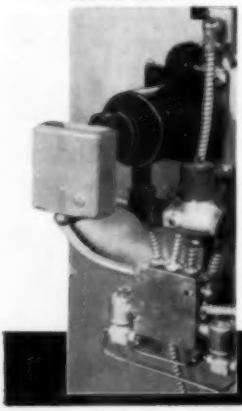

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ratio of court cases of malpractice to the number of physicians, or the ratio of number of court cases to years of practice, or the ratio of court cases to the number of medical cases treated is exceedingly low.

If it is true that there are areas today where there are more cases in litigation than the medical profession would like to see, it is also true that they are not numerically significant except to the medical profession.

With the information we have obtained from our public opinion survey and from our study thus far concerning professional liability, we can agree that good professional public relations can not be forced. It stems from the very concept of professionalism, a concept that applies to all professions. Your profession, my own, and medicine are all based on the concept of service.

Justice Shaw of the Supreme Court of Illinois expressed this concept clearly:

"The practice of a profession," he said, "is everywhere held to be subject to licensing and regulation under the police power and not subject to commercialization or exploitation. To practice a profession requires something more than financial ability to hire competent persons to do the actual work. It can be done only by a duly qualified human being and, to qualify, something more than mere knowledge or skill is essential. The qualifications include personal characteristics such as honesty, guided by an upright conscience, and a sense of loyalty to those served, even to the extent of sacrificing pecuniary profit, if necessary. The requirements are spoken of generically as that good moral character which is prerequisite to the licensing of any professional man.

Professional public relations encompasses tremendously more than a push button campaign of organizational advertising. Professions have enjoyed distinction and prestige throughout the years.

As society developed, the reputation of professions grew. Professional public relations essentially must consist of living up to the good reputation acquired by the professions through the years, and this good reputation did not just happen. It arose because the professions followed a standard of conduct much more stringent than the standards of conduct governing the actions of individuals and society.

This good reputation continued because the professions observed and enforced their own ethical standards. But, more importantly and most fortunately, in all the professions, there has always been a significant number of members who were and are sincerely dedicated to the principle of service rather than financial gain.

As a result, while the public has not exactly put the professions on a pedestal, the public expects the professions to act as if they were, and the professions must so act if they are to retain public respect and confidence.

Someone, I don't know who, said, "Our friends see only the best in us. For that very reason, they deserve the best from us."

Is the essence of good public relations, from a professional point of view, to be found in that statement? Do not the ethics, the traditions, the ideals of a profession compel those who comprise it to deserve the respect of the public because they have acted for the best interests of the public? And, when one accepts the prestige and the perquisites of professional life, must not he covenant with himself and his colleagues to accept the obligations of the profession which he chooses? Isn't it natural for the public to expect more from those whom it respects, and doesn't giving more induce more respect?

Professional public relations is based, then, on a careful and even sedulous observation of the spirit and intent of ethical principles of the profession by each member of the profession. It is nurtured by the continuing acceptance of obligations rightfully imposed upon the profession by itself and by the public.

Professional liability in medicine, when it is negligence, is a failure to discharge a duty to render proper care. It is the failure to discharge an obligation. If malpractice is medically inexcusable, or gross, or criminal, then the public may lose respect for the negligent one. And, depending upon many circumstances, the loss of respect for one may reflect against many.

The action, or the misaction, of one may affect good public relations, if that action is not recognized by the profession and if no steps are taken to insure the public that such action is not likely to happen again.

Isolated instances of malpractice so far seem not to have shaken the overall trust and confidence which the public has placed in the medical profession. This, I am sure, is undoubtedly due to the sum total of efforts of individuals and organizations to acquaint those dependent on medical service with the programs of the profession to improve its standards. Incidents of malpractice are exceptions which prove the

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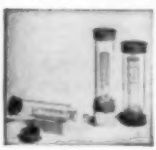

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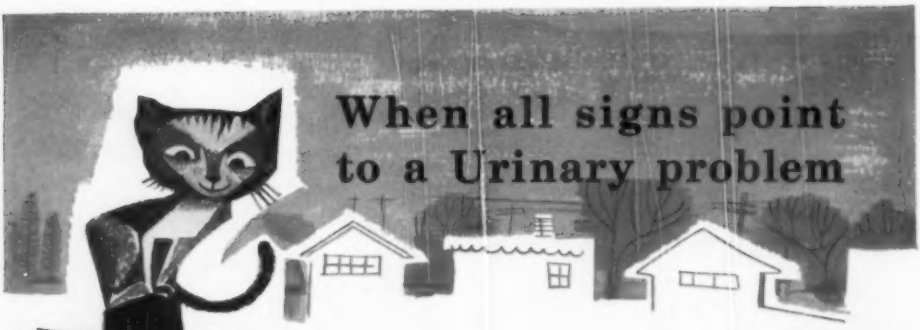

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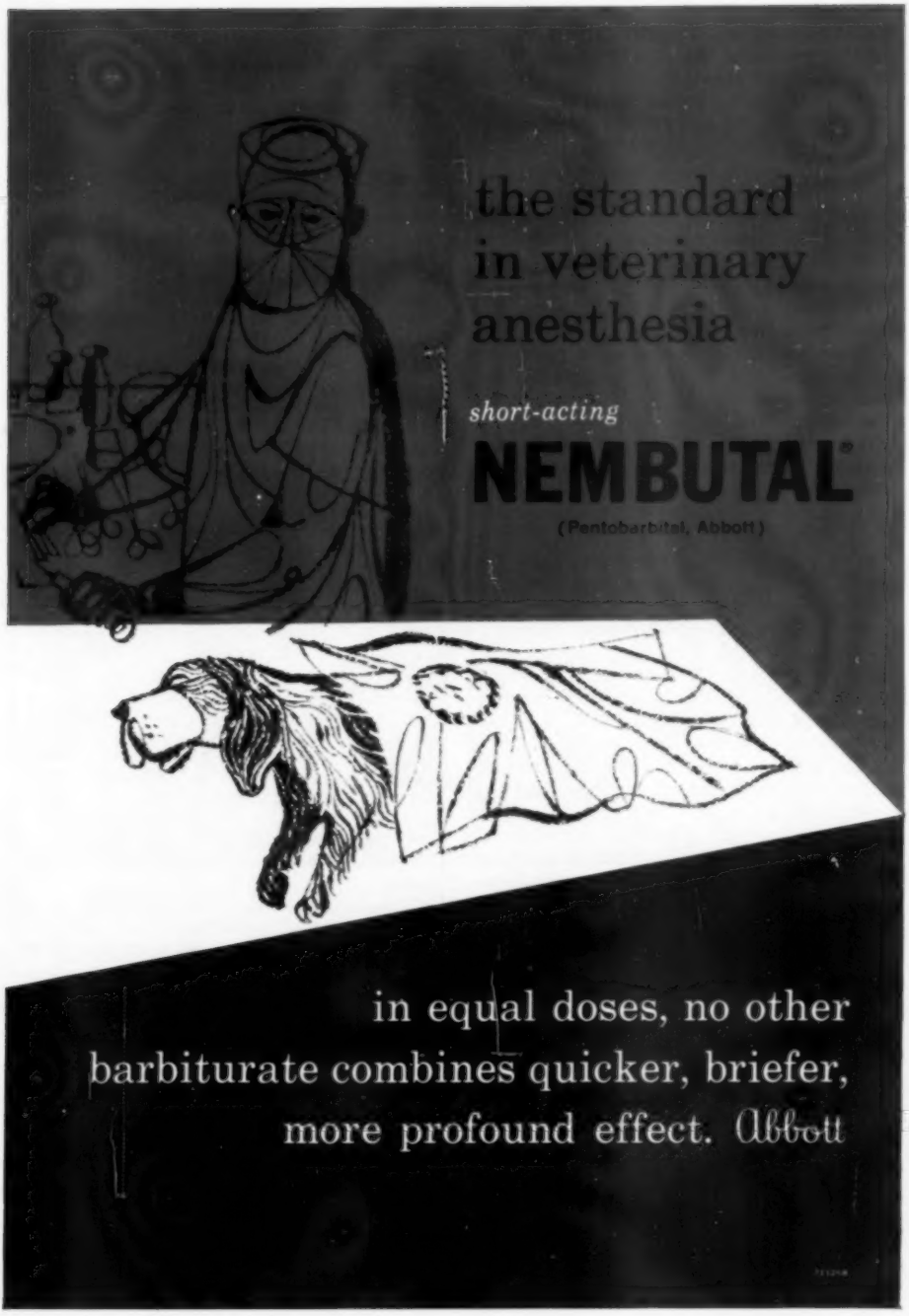
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Thank you.

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REFERENCES: 1. Bull, W. S.: N. Amer. Vet., In press. 2. Henry, R. T., and Blackburn, E. G.: Vet. Med., In press.

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*Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. 51:413 (Sept.) 1956.

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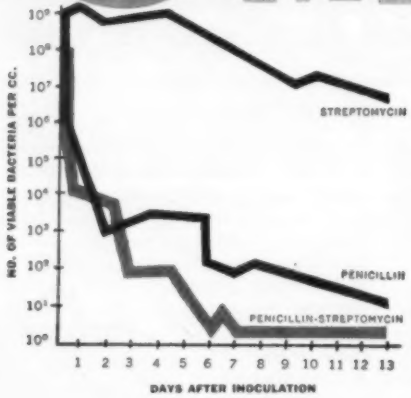
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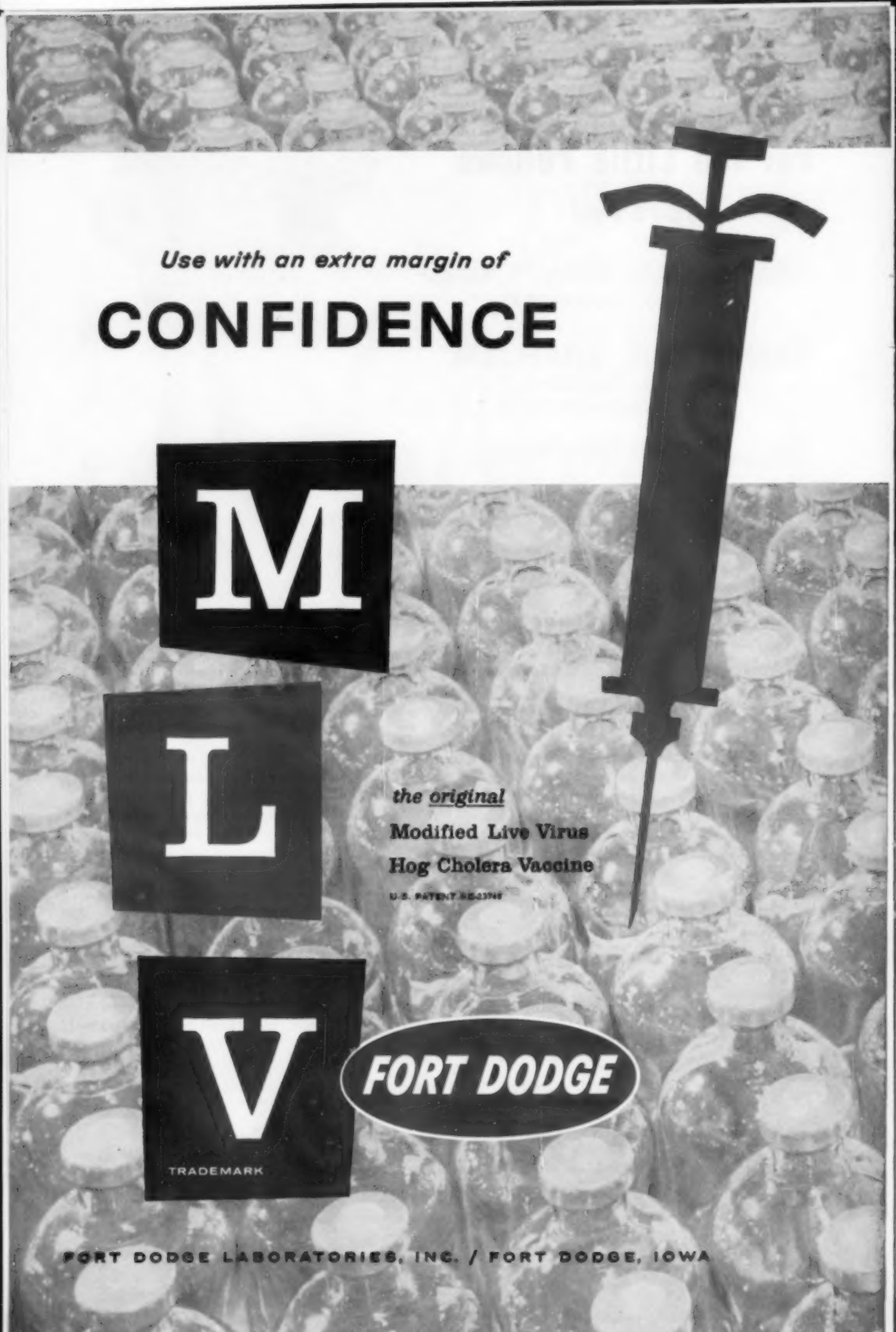
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Toxoplasmosis in the Cat—Fourteen Cases

HANS MEIER, D.V.M.; JEAN HOLZWORTH, D.V.M.;
ROBERT C. GRIFFITHS, D.V.M.

Boston, Massachusetts

WITHIN the past few years, evidence has accumulated that toxoplasmosis is more common both in man and in animals than had been realized. Widespread inapparent infection is suggested by serological tests, and infection coincidental with other diseases is encountered.

With growing interest in the cat, both as a pet and as a possible carrier of human disease, toxoplasmosis is being recognized increasingly in this species. The first report of feline infection described the presence of the protozoan in the lungs and mesenteric nodes of a 1-year-old cat acutely ill with inappetence, fever, cough, and mesenteric node enlargement.[12] Another instance of acute infection in a young cat was mentioned briefly in 1954,[7] and is presented here in more detail (table 1, case 3). Acute or subacute forms of the disease with effusion into the serous cavities have been observed in a kitten[14] and in a 2-year-old cat.[10] Intestinal granuloma has been reported in 2 aged cats,[5,10] and the organism has been found in association with chronic central nervous disturbances in 4 cats, 3 of which were recorded as adult or aged.[1, 7, 11, 10] Toxoplasma bodies have also been recognized in the brains of cats with more acute illness—1, age and symptoms unspecified, dying after 48 hours; the other, 2 years old, after a 10-day course characterized by irritability, incoordination, and disorientation.[3] Inapparent brain infection, represented by a few "pseudocysts"* unassociated with inflammatory reaction, was discovered in a cat belonging to a family in which there was a child with congenital toxoplasmosis. The cat's titer by the Sabin-Feldman serological dye test was 1:200.[2]

Although the group of published cases is small, it should not necessarily be concluded that toxoplasmosis is a rarity among cats. Many instances undoubtedly go unrecognized and still others that have been diagnosed remain unreported. Thus, several additional cases of acute infection in young cats have been brought to the attention of the authors by practitioners who suspected something out of the ordinary and sent tissues to their state laboratories or veterinary colleges.

It is hoped that, together with the published reports, a study of 14 more cases recognized at the Angell Memorial Animal Hospital in the years 1952 to 1956 will make possible some general conclusions as to the types of clinical disease with which Toxoplasma may be associated in the cat. In the 13 cases in which the disease ended fatally, the final diagnosis was made by histological examination. In 2 of these cats, the infection had been strongly suspected before death, and the diagnosis was confirmed in one instance by biopsy of a node, and in the other by the dye test. The latter cat's kitten, which became ill and recovered, was likewise shown by the dye test to have had toxoplasmosis.

Tissues were fixed in formalin, and sections were stained with hematoxylin-eosin and, in a few instances, with Giemsa's stain as well. In the case of chronic brain infection diagnosed at the Armed Forces Institute of Pathology, Barbeito-Lopez stain was also employed.

From these cases, details of which are tabulated

Dr. Meier, formerly acting pathologist at Angell Memorial Animal Hospital, is now a research associate at Children's Cancer Research Foundation and Harvard Medical School and an assistant pathologist at Children's Hospital, Boston. Drs. Holzworth and Griffiths are clinical staff members at the Angell Memorial Animal Hospital, Boston, Mass.

The authors thank Dr. G. E. Foley, of the Children's Medical Center, Boston, for making available the triazine compound used in the treatment of 2 cats, and Dr. Harry Feldman, of the College of Medicine of Syracuse University, for his performance of serological tests. They acknowledge the contribution of clinicians and pathologists, past and present, of the Angell Memorial Hospital, whose work is in any way represented in this study. They also thank Dr. Irwin Jungherr, University of Connecticut, and Major Charlie Barron, Armed Forces Institute of Pathology, for studying brain sections in 1 case of encephalitic infection.

Thanks are also due to Mr. Harold Marena, of the Angell Memorial Hospital, for his help in getting serum samples from the infected kitten, to Miss Kathleen Driscoll, x-ray technician at the hospital, for her fine radiographs, and to Mr. Leo Goodman, of Boston City Hospital, for his photographic work.

*This term is retained out of deference to tradition. Actually there is evidence that the structure possesses its own wall or membrane and is, therefore, genuinely a cyst.[9]

TABLE I—Clinical and Pathological Findings in 13 Fatal Cases of

Age, sex, date of admission	History, symptoms Diagnosis, outcome	Diagnostic procedures	Gross pathology	Histopathology
(1) Tiger 1 yr. Female 12/11/56	Course 5 da. Litter 2 mo. before; 1 weaned. Apathy, poor appetite, then dyspnea, thirst. Sl. jaundice, rales entire chest, tender abd., urine orange; t. 104.6 F. Diag. toxoplasmosis. Died in 24 hr.	12/11 XR diffuse lung density, esp. lower parts. w.b.c. 6,250, S 35%,* NS 33, L 27, M 4, E 1. Nucl. r.b.c. 13%, platelets decr. Hb. 14.5 Gm. ICT. INDEX elevated. PROTHROMBIN TIME incr. DYE TEST 1:4,096. URINE alb.+, bile+++. 12/12 w.b.c. 4,850, M 15%. Nucl. r.b.c. 30%, Hb. 12.5 Gm., hemolysis. MOUSE INOCULATION 5 hr. postmortem unsuccessful.	Lungs: edema, focal consolidation. Retrophar., mes. nn.†, enl. Liver, pale, mottled. Duodenum, ulcerated.	Lung impressions, toxopl. in macrophages. Bronchopneumonia. Focal myocardial degeneration. Acute hepatitis. Splenic hyperplasia. Lymphadenitis. Duodenal ulceration and granulation. Cellular infiltration medulla of brain. Marrow very active. Toxopl. all these tissues except brain, marrow.
(2) Gray long-haired 4 mo. Female 4/27/53	Course 5 da. Poor appetite. Cachexia, staggering, foul breath, pallor, sl. jaundice, dyspnea with bronchial sounds; t. 102.5 F. Diag. malnutrition, resp. complications. Died 3rd hosp. da.	4/27 XR diffuse lung density, esp. lower parts.	Poor cond., mod. jaundice. Lungs, pink-gray to brown, sl. enl., edematous, firm, purulent foci. Liver, light brown. Spleen, enl.	Severe bronchopneumonia, toxopl. Ac. hepatitis, passive congestion.
(3) Gray and white 1½ yr. Female 8/25/53	Course 9 da. Inappetence, thirst, vomited once. Dehydration, foul breath, intestinal (?) mass 4.5 x 1.5 cm.; t. 104.8 F. Diag. panleukopenia? intussusception? Died 3rd hosp. da.	8/26 XR fluid abd. cavity. w.b.c. 14,800, S 53%, NS 22, L 18, M 7. BIOPSY mes. n., ac. and chr. inflammation, toxopl. SKIN TEST neg. at 24 hr.	Pericard. and abd. fluid, incr. Mesentery, edematous. Mes. nn., enl., firm.	Ac. hepatitis; toxopl. Spleen reticular hyperplasia, follicles atrophied, necrotic and hyalinized; erythrophagocytosis; hemosiderosis; toxopl. Focal interstitial nephritis.
(4) White 2 yr. Male 12/24/55	Course 10 da. Apathy, inappetence, then dyspnea. Pallor, abd. resp., friction sounds whole chest; t. 103.0 F. Diag. pneumonia. Destroyed 3rd hosp. da.	12/27 XR irregular areas lung density, esp. lower parts.	Lungs, enl., edematous, gray-pink with white foci. Mediastinum, edematous, Mediast., mes. nn., enl. Spleen, enl.	Bronchopneumonia; toxopl. Subac. hepatitis, granulomas with toxopl. L. nn. hyperplasia, edema, necrosis; toxopl. Splenic hyperplasia.
(5) Gray and white long-haired 2 yr. Castrated male 11/18/52	Course 11 da. Panleukopenia 2½ mo. before. Thirst, lethargy, inappetence, retched once. Foul breath; t. 105.0 F. Later dyspnea, pallor, jaundice, terminal hyphemia, bleeding injection sites. Died 4th hosp. da.	11/18 XR not significant. 11/19 w.b.c 3,750.	Good cond., mod. jaundice. Free blood chest. Lungs, not collapsed, "meaty," reddened, subpleural hemorrhages, pale foci. Pericardium, bloody fluid. Liver, mottled yellow-brown; pale foci. Thor., mes. nn., enl., reddened.	Ac. bronchopneumonia; toxopl. Ac. hepatitis, chr. periportal inflammation; toxopl. Renal tubular biliary casts. Subac. meningo-encephalitis, perivascular demyelination, "pseudocysts" without reaction.
(6) Yellow 5 mo. Male 8/12/55	Fe. littermate had fatal pneumonia, leukopenia, monocytosis 8/10-8/19; Course in this cat 11 da. Apathy, anorexia, polypnea, enl. spleen; t. 105.0 F. Cont. high fever, progressive dyspnea. Died 10th hosp. da.	8/17 w.b.c. 2,450, S 44%, NS 4, L 48, E 4.	Fair cond. Lungs, firm, edematous, purplish, yellow mottling. Bronchial nn., reddened, swollen.	Ac. bronchopneumonia, focal necrosis; toxopl.
(7) Black and white 2 yr. Castrated male 10/11/55	Course actual illness 12 da. Poor appetite some time. Apathy, thirst, vomiting, dyspnea, cont. high fever. Comatose, pale, sl. cyanotic, exp. grunt, friction sounds; t. 106.0 F. Diag. ac. pancreatic or liver disease. Died 3rd hosp. da.	10/11 XR not significant. 10/12 w.b.c. 5,700, S 53%, NS 21, L 17, M 7, E 2. Nucl. r.b.c. 2%. Hb. 11.8 Gm. 10/13 URINE dark brown, alb. ++, bile +++, numerous r.b.c.	Sl. jaundice. Lungs, "meaty," dark red, edematous, necrotic areas. Liver, pale. Pancreas, bordered by necrotic fat.	Bronchopneumonia. Severe hepatitis. Splenic congestion, red pulp hyperplasia. Ac. to subac. necrotizing pancreatitis. Toxopl. all these tissues.

Toxoplasmosis. 1. Acute Systemic (Arranged in Order of Severity)

Age, sex, date of admission	History, symptoms Diagnosis, outcome	Diagnostic procedures	Gross pathology	Histopathology
(8) Red 4 mo. Male 12/27/55	Course 13 da. Anorexia, thirst, fever, dyspnea. Spirits, color good; rapid abd. resp; enl., tender liver; t. 104.0 F. Diag. lipoid pneumonia. Cont. fever; dyspnea severe. Destroyed 4th hosp. da.	12/28 XR dense spotty lung areas, esp. lower parts. w.b.c. 7,800, S 25%, NS 11, L 60, M 3, E 1. Platelets decr.	Lungs. enl., firm, red, edematous; pale areas coalescing in lower parts. Sternal, mes. nn., enl., pale, firm Liver, deep red, enl.	Bronchopneumonia; toxopl. Hepatic congestion, extramedullary hematopoiesis L.nn. reticular hyperplasia. Subac. pancreatitis; toxopl.
(9) Gray 6 mo. Male 1/9/54	Course 16 da. Appetite poor, diarrhea, episodes severe dyspnea. Thin apathetic; t. 104.5 F. No improv. though spirits, appetite good. Destroyed 8th hosp. da.	1/19 XR not significant. w.b.c. 26,000. STOOL numerous coccidia.	Cond. poor. Lungs, noncrepitant, congestion, emphysema, edema. Liver, finely mottled. Spleen, dark red, follicles prominent. L. nn., enl., pale brown, moist. Stool, bloody.	Bronchopneumonia; toxopl. Hepatic congestion, biliary scarring. Intestine, follicular hyperplasia, mucosa eroded, necrotic areas, mononuclear infiltration muscularis.
(10) White 1 yr. Castrated male 4/19/52	Course 1 mo. Anorexia, abd. "bloating," vomiting, given oil. Thin, dehydrated, abd. distended; t. 104.0 F. Ate, but cont. fever, incr. dyspnea. Destroyed 9th hosp. da.	4/21 w.b.c. 17,000. 4/27 THORACENTESIS small amount serosanguineous fluid.	Cond. poor. Peritoneal cavity, serous fluid. Lungs, dark, firm, bronchial mucopurulent exudate. Liver, enl., small superficial dark red nodules, some pale areas.	"Adenomatous" bronchopneumonia typical of toxopl. Liver, ac. centrolobular edema and hemorrhage, biliary scarring.
		2. Chronic Localized		
(11) Black 15 yr. Castrated male 3/2/55	Course final illness 12 da. Hosp. 2 mo. before, SQ lumbar abscess, thin, anemic, but spirits, appetite good. Gained till 10 da. before adm., then anorexia, vomiting diarrhea. Emaciated, dehydrated, pale; intestinal mass; t. 102.4 F. Later jaundice. Diag. lymphosarcoma? also pancreatis disease? Destroyed 3rd hosp. da.	3/3 w.b.c. 7,900, S 48%, NS 29, L 13, M 10, platelets decr. Hb. 6.6 Gm. URINE bile ++. STOOL foul, watery, gray curds; heavy coccidia, few fat droplets, no undigested starch or muscle.	Cond. poor. Liver, enl., mottled, yellow-brown, friable. Duodenum, reddened, edematous. Jejunum, papillary growths. Ileocolic junction, fibrous mass 3 x 4 x 3 cm.; necrosis serosal surface, hemorrhage, postnecrotic scarring mucosa. Colon, many small ulcers. Coccidia throughout intestine.	Hepatic congestion, centrolobular fatty degeneration, beginning cirrhosis. Jejunal papillomatosis. Ileocolic mass: granulomata submucosa and muscularis, encysted toxopl. among muscle fibers.
(12) White 13 yr. Castrated male 10/22/55	Course about 1 yr. Wt. loss 1 yr., anorexia, vomiting, diarrhea with blood 2 wk. Intestinal mass ant. abd.; t. 104.0 F. Diag. lymphosarcoma? Destroyed 3rd hosp. da.	10/22 XR segment small intestine thickened wall, dilated lumen. 10/24 w.b.c. 3,300, S 67%, NS 19, L 10, M 7, E 1, rare immature r.b.c., platelets decr. Hb. 7.2 Gm. URINE dark yellow, bile +. FECAL EXAM. neg.	Cond. poor. Pericardium, blood-tinged fluid. Liver, tan, rounded edges, distinct lobular pattern, many pale foci 2 mm. diam. Small intestine, several annular enlargements with dilated ulcerated lumen; 1 polypoid mass entending into lumen. Colon, ulcers. Adrenals, friable.	Focal hepatitis and necrosis, beginning cirrhosis; toxopl. Chronic granulomatous enteritis; ulcers; polyposis with toxopl. cysts. Mesenteric lymphadenitis, toxopl. Adrenal cortical necrosis; toxopl.
(13) Black and white 14 yr. Castrated male 4/30/54	Course 6 wk. Progr. post. weakness, hiding. Convulsion, then disturbed equilibrium. Distress, loud cries, rapid resp., unsteady gait; t. 101.0 F.		Cond. good. Liver, irregular distribution of blood. Spleen, follicular hyperplasia. Pancreas, yellow discoloration. Kidneys, fatty-looking. Brain, grossly normal.	Liver-cell vacuolation, edema, hemosiderosis. Splenic hemosiderosis. Pancreas fixation artefacts. Kidney fatty dystrophy prox. conv. tubules. Chr. diffuse meningo-encephalitis, toxopl. cysts; Encephalitozoon cerebral cortex, no inflammation.

S=segmented cells; NS=nonsegmented cells; L=lymphocytes; M=monocytes; E=eosinophils; nn=lymph nodes.

(table 1), it would appear that feline infections may be manifested either by acute or subacute systemic disease, or by chronic, localized lesions. With few exceptions, the two groups are sharply contrasted in respect to age incidence, duration and severity of illness, and type and dissemination of lesion.

ACUTE AND SUBACUTE SYSTEMIC INFECTION

Acute or subacute infections were recognized in 11 cats ranging in age from 9 weeks to 2 years. Seven of the cats were males, a proportion approximating that among ill cats generally at the Angell Memorial Hospital. Season did not appear to be a factor in incidence.

Signs.—At onset, the signs were nonspecific—lethargy, inappetence, weight loss, and often thirst, the latter perhaps attributable to the characteristic persistent high fever. A later and more specific change was a progressively severe dyspnea, apparently a common feature of acute infections, since it was observed sooner or later in a majority of the 11 cats. Respirations were of a deep, abdominal type and of striking rapidity. In contrast with the dyspnea occurring with conditions in which fluid fills the thorax, loud friction or bronchial sounds were heard over the entire chest. None of the cats coughed.

In several cats, the signs were those of severe systemic disease or of an acute abdominal disorder—panleukopenia, hepatitis, pancreatitis, or intestinal obstruction by intussusception or foreign body. Prostration, occasional vomiting, abdominal tenderness, liver enlargement, mild jaundice, and bilirubinuria characterized this type of case. Pallor, but not of severe degree, was observed in 3 cats on admission, and it developed in another, in which blood appeared just before death in the anterior chambers of the eyes and in which there was bleeding from injection sites. In the one cat in which a presumptive diagnosis was made on admission (table 1, case 1), the combination of the characteristic rapid respirations and pneumonic sounds with mild jaundice and marked bilirubinuria was considered virtually pathognomonic.

Diagnostic Procedures.—Since the prominent sign was usually respiratory or abdominal distress, a radiograph was in most cases taken on admission.

Because of the rapidity of respirations, it was impossible, with the 1/8-second exposure time required by the machine in use through 1955, to obtain pictures entirely free of blurring due to motion. With a machine permitting an exposure of 1/20 second, satisfactory pictures were achieved. In cats in which respiratory difficulty was just beginning, no abnormal lung shadows could be detected. When dyspnea had become pronounced, spotty areas of increased density appeared in the lungs, particularly in the lower parts (fig. 1, 2). These radio-

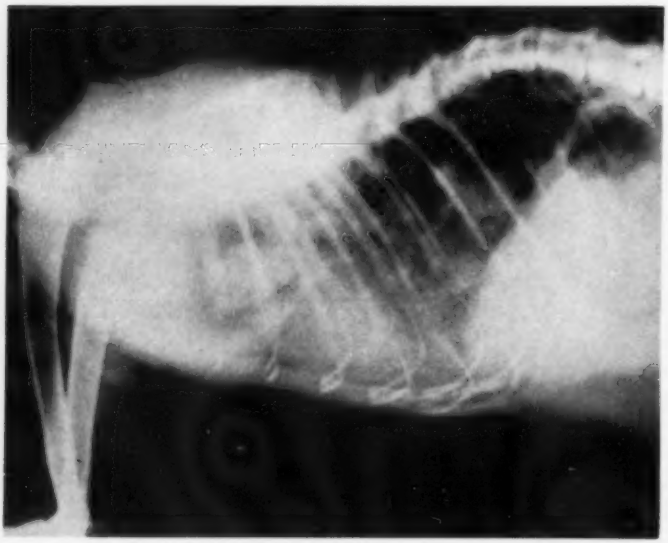

Fig. 1—Lateral chest radiograph of a cat with severe dyspnea due to acute toxoplasmosis. Because of the rapidity of respirations it is difficult to avoid blurring with an exposure of 1/8 second. Dense, spotty shadows in the apical lobes and the lower parts of the diaphragmatic lobes are typical of pulmonary toxoplasmosis, but also occur in inhalation pneumonia.

graphic features, strongly suggestive of foreign-body pneumonia, in several instances gave rise to the mistaken impression that the condition was due to inhalation of mineral oil, a remedy popular with cat owners and actually given to some of these patients.

In 2 cats in which the signs suggested an acute abdominal condition, peritoneal effusion obscured the picture. In 1, a palpable abdominal mass proved at exploratory operation to be greatly swollen mesenteric nodes. On suspicion of toxoplasmosis, one was removed for examination. Although it is known that skin sensitivity is not usually present in acute infections, a diagnostic injection of toxoplasmin-histoplasmin was given, but the cat died about 24 hours later, before sufficient time could elapse for evaluation of the response to the test. The lymph node removed for biopsy proved

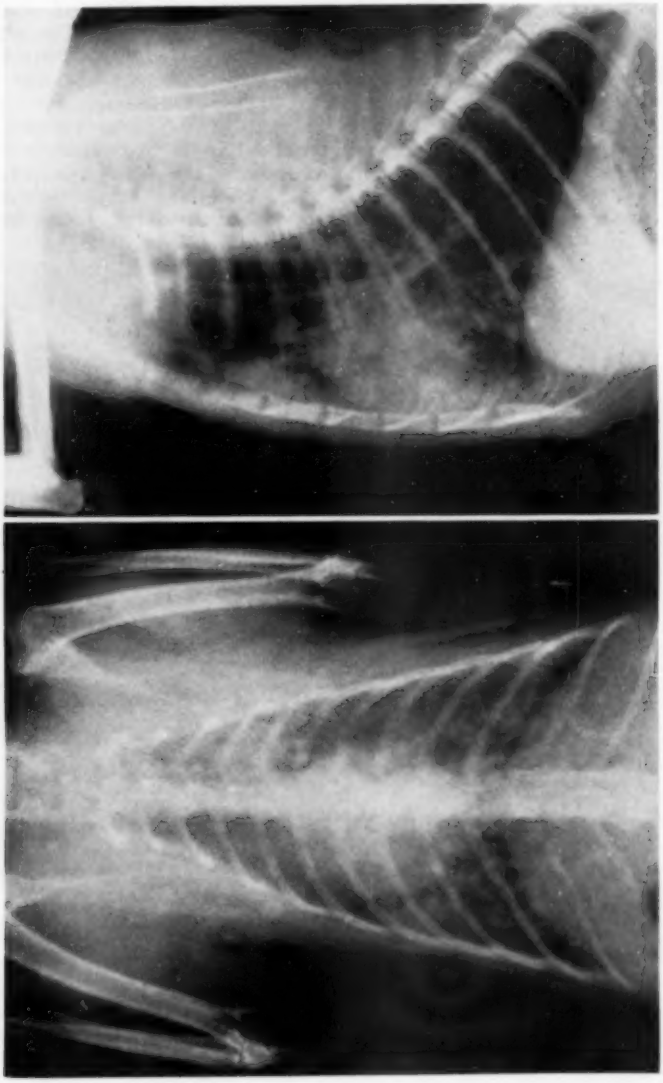

Fig. 2—Lateral and dorsoventral chest radiographs, showing acute toxoplasmal bronchopneumonia. Changes are less severe than in figure 1, but the areas of density are similarly distributed. Involvement of the apical lobes is evident. Exposure 1/20 second.

to contain macrophages with Toxoplasma bodies.

Blood studies were usually made at or shortly after admission, and indicate what may be expected when infection is well established. In total leukocyte counts in the acute and subacute cases, moderate leukocytosis and leukopenia were both represented (range 26,000—2,450 w.b.c./cmm.), but there is a strong suggestion that, as the infection progresses, the tendency is toward the latter. In differential leukocyte counts (6 cases, 13 counts), even when leukopenia was present, neutrophils usually outnumbered lymphocytes, and in some cats monocytes were significantly increased. In several, platelets were diminished.

Severe anemia did not appear to be prominent at the time of admission, since in most instances pallor was not sufficiently striking to prompt the performance of hemoglobin determinations. Color of the mucous membranes suggested, however, that anemia developed terminally in cats with extensive organic involvement.

Blood changes in the case of the sole survivor are tabulated for the course of the illness (table 2). The first hemogram and the series made after recovery suggested that in this kitten lymphocytes perhaps habitually outnumbered neutrophils. With the development of infection, the total leukocyte count gradually fell, although the ratio of neutrophils to lymphocytes rose. The production of platelets and of erythrocytes also decreased. While these changes may well have been the result of the disease, the folic acid antagonists (1,2-dihydro-*s*-triazines and pyrimethamine) given in treatment must not be overlooked as possible contributing factors. Indeed, when the platelets almost disappeared, the leukocyte count fell to 3,000 per cubic mil-

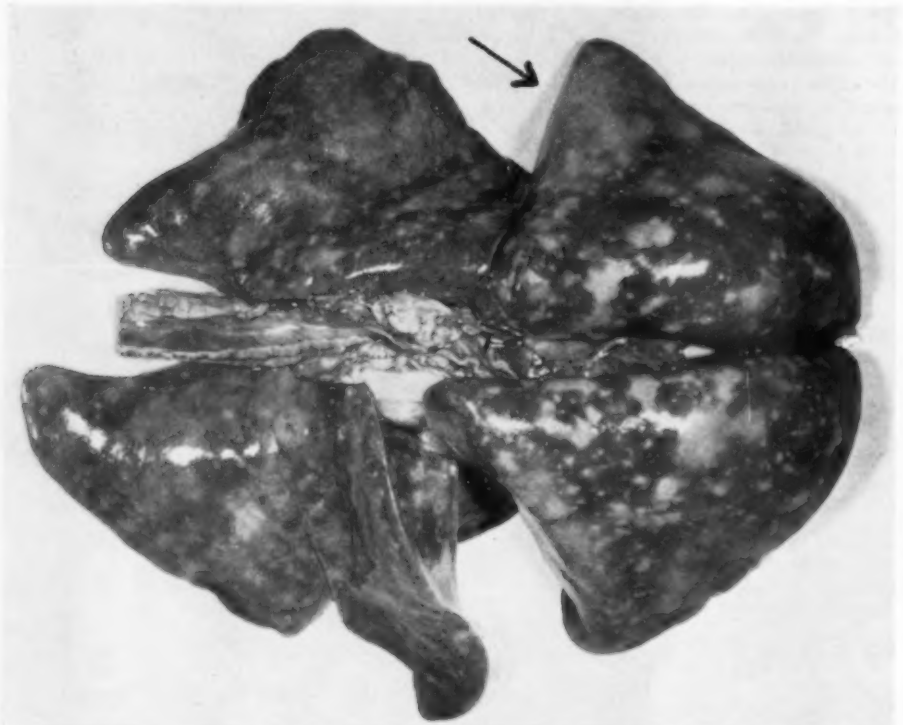

Fig. 3—Bronchopneumonia due to toxoplasmosis, dorsal view. The appearance of the lungs resembles that in foreign-body pneumonia. They do not collapse normally and are congested, with firm, irregular, light areas. These changes, although diffuse, are pronounced at the lower angle of the right diaphragmatic lobe, where consolidation is conspicuous (arrow).

limeter, and the hemoglobin began to drop, it was considered advisable to give folic acid. Whether or not the folic acid alone was responsible, its administration was followed by a sharp rise in both leukocytes and hemoglobin.

Although 13 blood films from 6 cats were thoroughly examined, Toxoplasma bodies were conclusively identified in none.

Serological diagnoses by means of the dye test were made in 2 cats. Serum samples obtained from 1 on the fifth day of illness and a day later at the time of death showed titers of 1:4,096 (case 1). The successive antibody levels in this cat's kitten, which developed and recovered from the disease, are recorded in table 2. Five months after recovery, as at the height of illness, her titer was 1:1,024.

Treatment.—Treatment included a variety of antibiotics,* B-complex vitamins, subcutaneous administration of fluids to overcome or prevent dehydration, and oxygen whenever dyspnea became severe. The absence of response to antibiotics was constant. In the cat in which the condition was tentatively diagnosed on admission (case 1), treatment was started with a triple sulfonamide and 1,2-dihydro-*s*-triazines, a combination exhibiting synergistic effectiveness in experimental murine toxoplasmosis,[17] but death occurred in less than 24 hours.

This cat's kitten, as soon as it developed a fever, was assumed to have toxoplasmosis and was accordingly treated at once with a sulfonamide. When a transient drop in fever was followed by another rise, administration of triazines was started by subcutaneous injection. A severe skin reaction developed, however, and pyrimethamine (Daraprim†), a folic acid antagonist apparently curative in several human cases of systemic toxoplasmosis,[4,18] was substituted. Dosages of these drugs and the correlation between administration and clinical signs are indicated (table 2). It is impossible to assert positively that these two drugs, which were administered together with sulfonamides, with which they appear to act synergistically,[4,17,18] favorably affected the course of the disease, but there is no question that immediate and significant clinical improvement coincided with their use; and when pyrimethamine was repeated in two subsequent febrile episodes, the same apparent effects were observed.

Course of the Disease.—The course of illness varied in the fatal cases from less than one to two weeks, being more rapid and severe in the cats in which involvement was more extensive. The 5 cats most acutely affected died within one to four days of admission to the hospital. Two became so dyspneic that they were destroyed to prevent needless suffering. In 2 others, 1 of which continued in good spirits and even ate, the course might have been considerably prolonged, but they were destroyed because of persistent fever and continued or increasing respiratory distress.

In the kitten that was treated and survived, the duration of illness from the first temperature rise to apparently complete clinical recovery was about four weeks. An initial episode of high fever and inappetence was followed by marked improvement coincident with administration of the triazines and pyrimethamine. Possibly because these were not continued long enough, there soon ensued a relatively prolonged period of low-grade fever, poor appetite, and diarrhea characterized by a clay-colored, greasy stool. During this time, both pyrimethamine and sulfonamides were resumed, and fluids, B-complex vitamins, folic acid, and bismuth were given as supportive measures.

Epidemiology.—As already indicated, 2 of the affected cats were a mother and her kitten. Of great interest in the history of another cat (case 6) is the fact that its littermate also almost certainly died of toxoplasmosis. The latter, a female, was hospitalized two days before the other, with identical signs of lethargy, inappetence, and progressively severe pneumonia. The total leukocyte count on admission was 9,500 per cubic millimeter, but six days later had fallen to 450 (neutrophils 60%, monocytes 10%, lymphocytes 30%). On the tenth hospital day, the cat died in intense respiratory difficulty, but unfortunately did not come to necropsy.

For what it is worth, background information on still another cat (case 3) may be mentioned. This animal belonged to an apartment house janitor who always kept

*Penicillin-streptomycin; chlortetracycline (Aureomycin, American Cyanamid Co., Pearl River, N.Y.); oxytetracycline (Terramycin, Chas. Pfizer and Co., Inc., Brooklyn, N.Y.); chloramphenicol (Chloromycetin, Parke, Davis and Co., Detroit, Mich.).

†Daraprim, the trade name for pyrimethamine, is produced by Burroughs Wellcome and Co., Tuckahoe, N.Y.

TABLE 2—Course of Illness in a 9-Week-Old Female Kitten Naturally Infected with Toxoplasmosis

Date	Clinical data	Temperature	Laboratory findings	Serum titer by dye test	Treatment
12/13/56	Adm. in good health day after mother died of ac. toxopl.	101.0 F.	Fecal exam. neg.	No antibodies	Anti-panleukopenia serum; repeated every 2 wk.
12/17	Loose stool.	103.5 F.	....	....	0.125 Gm. sulfadiazine b.i.d.
12/18-19	Stool normal.	102.5 F. a.m. 103.2 F. p.m. 12/19, 102.0 F.	12/18 w.b.c. 10,500, S 29%, NS 5, L sm. 41, L int. 18, L lg. 2, M 5, occas. basophilic r.b.c., some anisocytosis. Hb. 11.7 Gm.	12/18 1:256	Same
12/20	Lethargy, poor appetite.	103.8 F. a.m. 104.6 F. p.m.	....	....	Same, plus 0.5 cc. suspension of 1,2-dihydro-s-triazines (50 mg. D 63, 20 mg. D 110 in 10 % Tween 80) SQ.
12/21	Brighter, but anorexia. Severe reaction at site of triazine injection.	103.9 F. a.m. 103.3 F. p.m.	....	....	Sulfadiazine continued, triazines stopped, 6.25 mg. pyrimethamine (Daraprim) orally.
12/22-23	Much improved, still inappetent.	range 102.6 – 101.6 F.	12/22 w.b.c. 8,650, S 55% NS 8, L sm. 15, L int. 17, L lg. 2, M 3, decr. platelets. Hb. 10.6 Gm.	1:1,024	Sulfadiazine continued; 5% dextrose, amino acids, and B complex SQ.
12/24-25	Ate a little, 12/25, no appetite.	101.6 F. 102.0 F.	....	....	Sulfadiazine stopped, fluids. B complex continued.
12/26	No appetite, depression, stool loose and light, sl. dehydration.	102.0 F.	....	....	Ringer's solution, amino acids, B complex SQ. 6.25 mg. pyrimethamine, 0.125 Gm. triple sulfonamide b.i.d., milk of bismuth, hand feeding.
12/27	Brighter, ate a little, stool loose, brown.	102.3 F.	S 59%, NS 13, L sm. 28, L int. 3, M 4, E 1, nucl. r.b.c. 1%, rare basophilic r.b.c., platelets sl. incr., v. small.	....	Same, pyrimethamine 2.5 mg.
12/28	Continued improvement, but appetite still poor, stool soft-loose.	102.2 F.	....	1:1,024	Same.
12/29-31	Bright, appetite poor to fair; stool gray, loose, becoming soft.	102.7 F. to 102.0 F.	12/29 w.b.c. 3,150, S 13%, NS 2, L sm. 50, L int. 33, M 2, platelets a little larger. Hb. 8.8 Gm.	....	Same.
1/1/57	Appetite good, no stool.	102 F.	....	....	Previous treatment stopped; started 2.5 mg. folic acid orally.
1/2-4	Good appetite and spirits, normal stool.	1/2 103.0 F. a.m. 103.6 F. p.m. 1/3 and 1/4: 102.5 F.	1/2 w.b.c. 3,350, S 7%, NS 11, L sm. 43, L int. 18, L lg. 5, M 13, E 3, platelets still decr. and small, a few nucl. and basophilic r.b.c. Hb. 7.9 Gm.	....	Same.
1/5-7	1/5 stool soft, grayish 1/6-7 stool firm, dark gray.	102.1 to 102.5 F.	1/5 w.b.c. 24,000, S 41%, NS 26, L sm. 16, L int. 6, M 12, nucl. r.b.c. 4% platelets incr. Hb. 6.6 Gm.	....	Treatment stopped.

TABLE 2 (continued)—Course of Illness in a 9-Week-Old Female Kitten Naturally Infected with Toxoplasmosis

Date	Clinical data	Temperature	Laboratory findings	Serum titer by dye test	Treatment
1/8	Lessened appetite, stool normal.	102.8 F. a.m. 103.3 F. p.m.	w.b.c. 27, 800, S 53%, NS 11, L sm. 16, L int. 8, M 7, E 5, rare basophilic r.b.c., platelets incr. Hb. 9.6 Gm.	— — —	2.5 mg. pyrimethamine; 0.125 Gm. triple sulfonamide b.i.d.
1/9-15	Appetite fair, then good, stool variably soft or loose.	Fluctuated between 102.5 and 101.8 F.	— — —	1/9 1:024	Same. 1/14 2.5 mg. folic acid resumed.
1/16	Stool normal.	101.4 F.	w.b.c. 17,550, S 35%, NS 9, L sm. 38, L int. 11, M 7, E 1, platelets very num. Hb. 10.6 Gm. PROTHROMBIN TIME 12.9 sec. (normal).	— — —	Same, plus milk of bismuth.
1/17	Good health	101.8 F.	— — —	— — —	Treatment stopped.
1/23	Continued good health.	101.6 F.	w.b.c. 16,700, S 44%, NS 7, L sm. 30, L int. 10, M 3, E 3, rare basophilic r.b.c. Hb. 11.7 Gm.	— — —	None.
1/30	Continued good health.	101.6 F.	w.b.c. 14,200, S 25%, NS 7, L sm. 45, L int. 8, M 1, E 6. Hb. 11.3 Gm.	— — —	None.
2/15	Continued good health.	102.0 F.	w.b.c. 15,200, S 27%, NS 5, L sm. 56, L int. 2, M 6, E 4. Hb. 12.4 Gm.	— — —	None.
2/27	Continued good health.	101.0 F.	w.b.c. 12,100, S 26%, NS 5, L sm. 33, L int. 25, M 5, E 6. Hb. 11.0 Gm.	1:1,024	None.
3/15	Continued good health.	101.0 F.	w.b.c. 11,250, S 28%, NS 5, L sm. 47, L int. 4, L lg. 1, M 7, E 8. Hb. 11.7 Gm.	— — —	None.
6/1	Continued good health.	101.8 F.	— — —	1:1,024	None.

a number of cats as mousers. He had previously lost cats with swift, severe illnesses of this general type, presumed at the time to be panleukopenia. Other cats had died with more protracted disorders characterized by central nervous manifestations.

Gross Pathology.—Loss in condition was, in general, significant only in the more prolonged illnesses. Mild jaundice was present in several cats.

The lungs had a similar appearance from case to case (fig. 3). They failed to collapse normally, were reddened, with irregular, light spots, were of "rubbery" consistency, particularly in the dependent portions, and on sectioning bulged and oozed a yellowish fluid. Rapid differentiation from lipoid pneumonia was achieved in some instances by the use of imprints stained with Sudan IV. In the cat in which the condition was diagnosed clinically, tentative confirmation was given at the time of necropsy by lung impressions stained with Wright's and Giemsa's stains and showing macrophages containing bodies highly suggestive of Toxoplasma (fig. 4).

Liver changes were not striking, although moderate swelling or friability was sometimes present, as well as abnormalities in color, such as pallor, mottling, or focal yellowish areas of necrosis. The spleens

were sometimes moderately enlarged, and there was variable enlargement and reddening of the thoracic and abdominal nodes. In one striking, peracute infection with extensive organic involvement, the pancreas was the site of the principal gross changes, being surrounded with areas of necrotic fat. In another case, there were intestinal ulcers. Effusion into the pericardial, thoracic, and peritoneal cavities was present in several cats, but not in great quantity. In 1, the fluid contained blood.

Histopathology.—The histological picture in all lung sections was constant and in accord with the findings of others that there is a type of bronchopneumonia highly characteristic of Toxoplasma infection. A tremendous hyperplasia and hypertrophy of the alveolar and bronchial lining cells and a fairly broad, edematous interstitium combined to present an "adenomatous" pattern, somewhat resembling that in adenomatosis of cattle and sheep. Desquamated lining cells, macrophages, and neutrophils filled the alveolar spaces, and macrophages and neutrophils infiltrated the interstitium (fig. 5, left). Numerous Toxoplasma bodies were present, packed into macrophages and alveolar lining cells (fig. 5, right) or freed by rupture of the cell membranes. In the more acute cases, there were disseminated foci of necrosis.

The heart muscle, sectioned in 1 case, had undergone interstitial inflammatory infiltration, and macrophages containing Toxoplasma were present.

In 6 cats, there was acute or subacute hepatitis, manifested by varying degrees of round cell infiltration, focal disseminated necrosis, and centrolobular hemosiderosis and atrophy. The Kupffer cells, projecting prominently into the sinuses, frequently contained Toxoplasma bodies, either single or in clusters (fig. 6, left). Less often, the liver cells themselves were parasitized (fig. 6, right), with resulting karyorrhexis and pyknosis. The cord architecture was sometimes disrupted by congestion and edema. Several of the livers

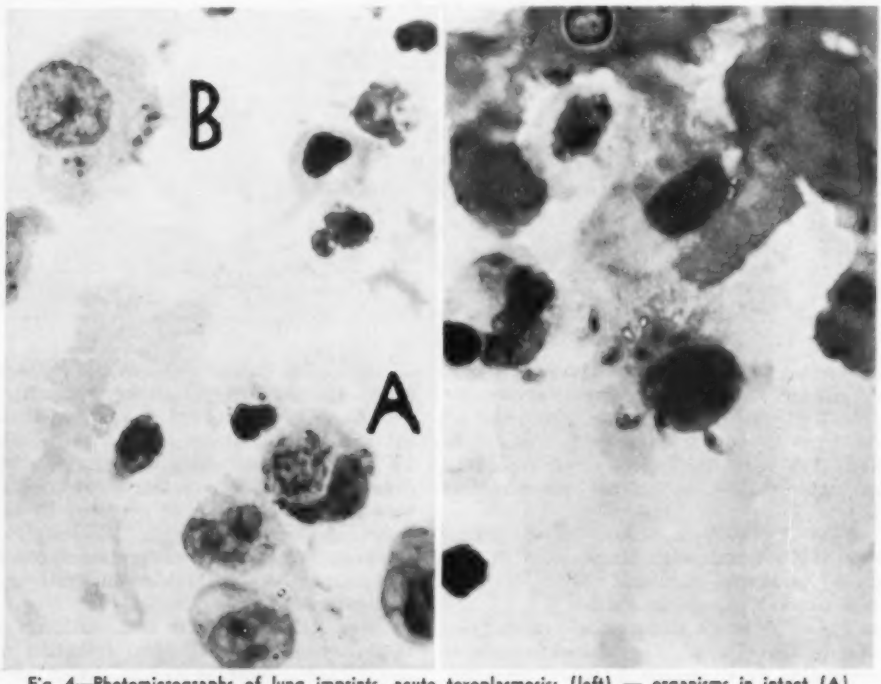

Fig. 4—Photomicrographs of lung imprints, acute toxoplasmosis: (left) — organisms in intact (A) and disrupted (B) macrophages, x 900; (right) — Toxoplasma organisms clustered about the nucleus of a macrophage (center). When viewed lengthwise, they are banana-shaped. The central body stains reddish purple and the cytoplasm blue-gray with Giemsa's stain. x 1,200.

underwent periportal myeloid metaplasia. In 3 cats, there was evidence of preexisting cholangitis.

In the case of extensive peripheral pancreatic fat necrosis, the parenchyma of this organ was obliterated by necrosis, and Toxoplasma bodies were numerous in the acinar and ductal epithelium (fig. 7, left). In another cat, there was a subacute pancreatic inflammation, with numerous Toxoplasma organisms clustered within cells or free in the interstitium.

In the cat with intestinal ulcerations, there were organisms not only in the cells of the muscular layer but also in the mucosal cells and interstitium (fig. 7, right).

No parasites were recognized in the few kidneys examined.

In the 5 cases in which the spleen was sectioned, this organ had undergone reticular hyperplasia, with atrophy of the lymph follicles, and the sinuses were filled with macrophages, in some of which were aggregated or single Toxoplasma bodies. In one instance, the follicles showed central necrobiosis and deposition of hyaline material, and there were slight erythrophagocytosis and hemosiderosis, interpreted as being a result of stress.

Lymph nodes were characterized by reticular hyperplasia, with acute or subacute inflammation of both parenchyma and perinodal tissue. Single or massed Toxoplasma organisms occurred in macrophages and lining cells of the sinuses.

In 1 of 2 cats in which the brain was examined, subacute meningo-encephalitis and perivascular demyelination were found. Toxoplasma "pseudocysts" were observed in the gray and white matter, but were unaccompanied by pathological reaction. Except for what had been considered a terminal "disorientation," there had been no signs of brain involvement. In the other cat, there were focal aggregations of macrophages in the brain substance and around small vessels, but no organisms could be discovered.

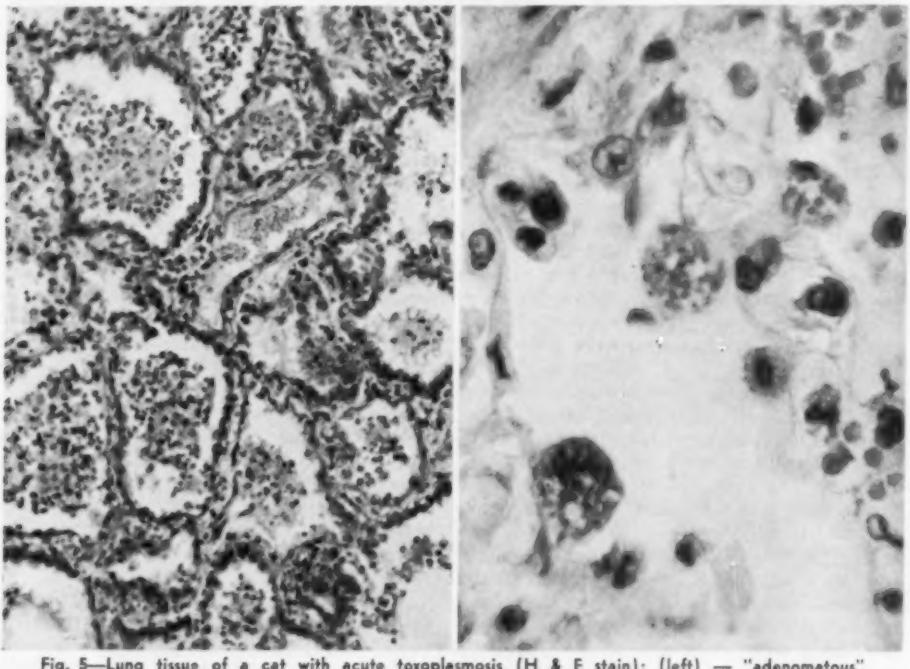

Fig. 5—Lung tissue of a cat with acute toxoplasmosis (H & E stain): (left) — "adenomatous" bronchopneumonia — the alveolar and bronchial lining cells are hyperplastic and hypertrophied, the interstitium is edematous, and there are acute cellular exudation and desquamation, x 200; (right) — hypertrophied alveolar cells contain groups of organisms (lower left, center, upper right). x 1,100.

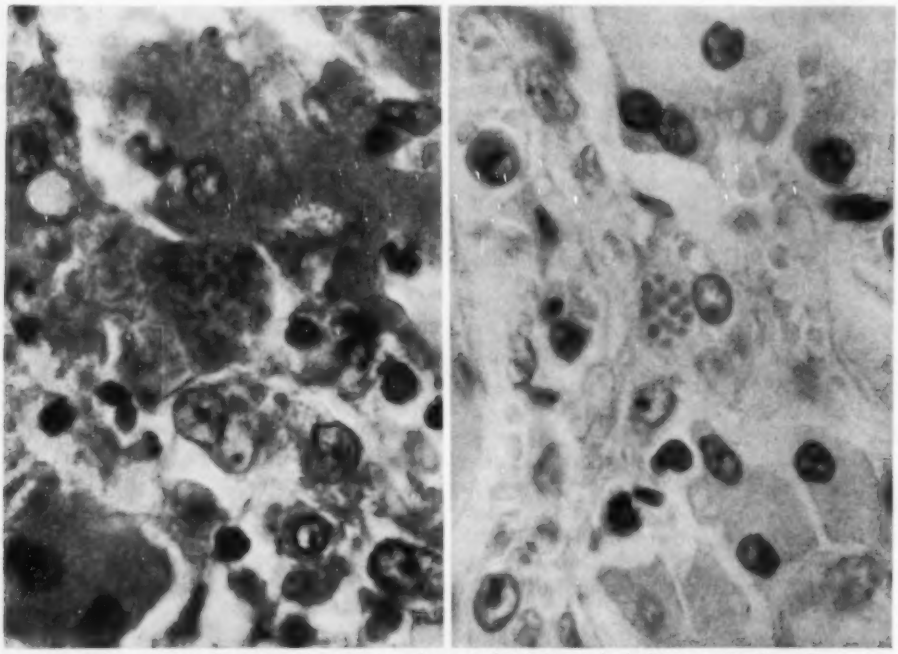

Fig. 6—Liver tissue of a cat with acute toxoplasmosis (H & E stain): (left) — a Kupffer cell projecting into a sinus contains Toxoplasma bodies (center), x 1,500; (right) — less often the organisms (center) appear in the parenchymal cells, x 1,250.

CHRONIC INFECTIONS

ENTERIC INFECTION

Signs.—Infection of chronic type was encountered in 2 aged, castrated male cats with relatively long histories of weight loss and diarrhea. Their final, rapid decline was associated with vomiting of all food, owing to obstructive masses in the intestine. The temperatures on admission were 102.4 and 104.0 F. One's stool was gray, foul-smelling, and curdy, suggesting pancreatic disease; however, microscopic examination disclosed no undigested fat, starch, or meat fibers, but did reveal a heavy coccidial infection. The other's stool was watery brown with traces of blood but no evidence of parasitism.

One cat had a normal leukocyte count, the other cat was leukopenic; in both there was neutrophilia. In neither of the blood films were Toxoplasma bodies observed. Both cats were anemic, probably owing to deprivation of food as well as to the presence of chronic infection, and their urine contained a small amount of bile pigment. In both cats palpable, firm, elongated enlargements in the intestine suggested tumor, intussusception, or foreign body. A radiograph in one instance revealed the annular mural thickening and narrowed lumen frequently seen in lymphosarcoma (fig. 8, top). Surgery was contemplated for both cats and supportive treatment started, but, because of their continued decline and the development of jaundice, it was considered best to destroy them.

Gross Pathology.—No abnormalities were observed in the thoracic organs. The livers were slightly swollen; one was mottled, brownish yellow, the other had many small, light yellow areas on its surface and a pale nodule extending into the parenchyma. The spleens and all lymph nodes appeared normal.

Major changes were limited to the intestine. In one cat a pale, fibrous mass involved the ileocolic junction; its mucosal surface was ulcerated and necrotic, with beginning scarring (fig. 8, bottom). Protruding from the jejunal mucosa were nu-

merous small, papillary growths. The mucosa of the colon was ulcerated. Scrapings revealed coccidia throughout the intestine as high as the duodenum.

In the other cat, there were several firm, annular thickenings in the wall of the small intestine, and the lumen of these segments was ulcerated. From one mass, a polyp-like protrusion extended into the lumen. Ulcerations occurred in the terminal portion of the colon.

Histopathology.—In one cat, the tumor-like mass involving the ileocolic junction proved to be a granuloma with occasional Toxoplasma "pseudocysts" among the muscle fibers (fig. 10). In the jejunum there was papillomatosis, but no Toxoplasma bodies were recognized. The changes in the liver were characteristic of circulatory disturbance—congestion, edema, centrolobular atrophy, and beginning cirrhosis. The spleen had undergone reticular hyperplasia.

In the other cat, there was wider dissemination of Toxoplasma organisms. Throughout the involved segments of the intestine there was chronic granulomatous enteritis with ulceration and focal polyposis associated with the presence of Toxoplasma "pseudocysts." Liver sections showed a focal hepatitis with necrosis and connective tissue proliferation. Toxoplasmata were present in macrophages and Kupffer cells. The spleen was normal, but in the mesenteric node there was inflammatory reaction to Toxoplasma bodies. Focal necrosis in the adrenals was also associated with these organisms. The presence of acute lesions in the liver, lymph nodes, and adrenals, together with a lesion of long-standing in the intestine, may conceivably be evidence for a recent flare-up of an earlier infection.[5]

CENTRAL NERVOUS SYSTEM

In one aged cat, the signs of illness—progressive incoordination and terminal convulsions—were wholly those of chronic brain disturbance, and toxoplasmosis was among the possible causes considered.

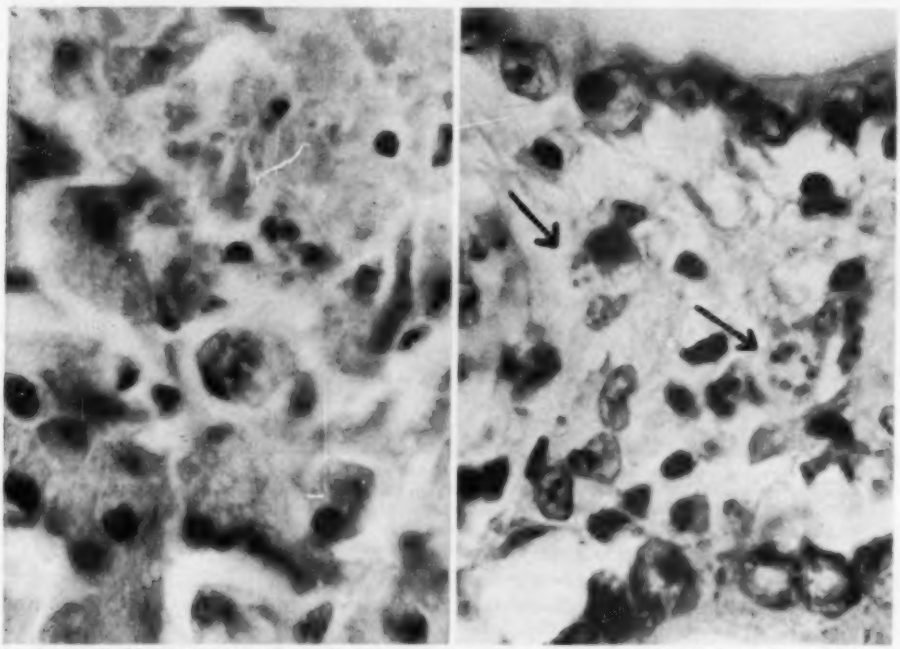

Fig. 7 (Left)—Necrotizing pancreatitis in acute toxoplasmosis. An alveolar cell (center) contains a group of organisms. In the necrotic area to the right above, scattered, free toxoplasmata are barely discernible as dark dots. H & E stain; x 1,250.

(Right)—Small intestine, acute toxoplasmosis. Organisms (arrows) taken up by macrophages in an edematous interstitium between glandular crypts. H & E stain; x 1,250.

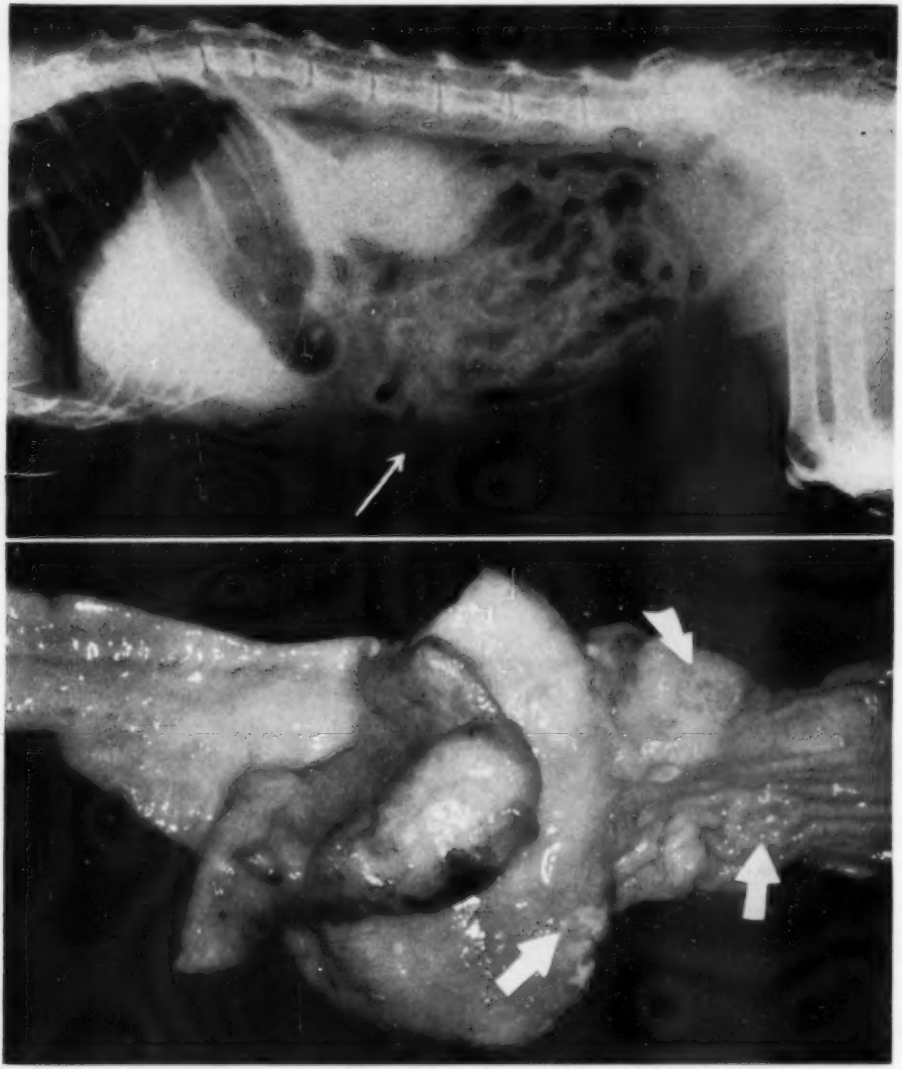

Fig. 8 (Top)—Lateral abdominal radiograph of a cat with chronic intestinal toxoplasmosis. Thickening of the wall of a segment of small intestine is recognizable in the lower abdomen (arrow). Radiographically, such a granuloma is indistinguishable from an annular lymphosarcoma.

(Bottom)—Toxoplasma granuloma, ileocolic junction. The intestine has been opened to reveal a pale, tumor-like enlargement. There are superficial ulcerations (arrows) of the colon (right).

Some of the brain sections* disclosed a chronic, diffuse meningo-encephalitis, the origin of which was not clear, and, scattered throughout the cerebrum, cystic structures containing bodies suggestive of Toxoplasma. In other sections,† there were typical Toxoplasma "pseudocysts" (fig. 10), as well as structures suggestive of Encephalitozoon, an organism described principally in rodents but also in a case of

*Studied by Major Charlie Barron, Armed Forces Institute of Pathology (Acc. 675249).

†Examined by Dr. Irwin Jungherr, University of Connecticut, Storrs.

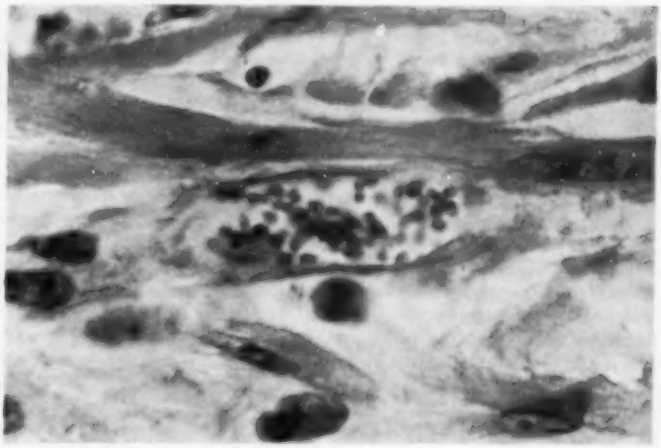

Fig. 9—Granuloma of ileocolic region with "pseudocyst" (center) embedded in proliferating tissue. H & E stain; x 1,500.

spontaneous meningo-encephalitis in a cat.[13]

DISCUSSION AND CONCLUSIONS

The 12 cases of feline toxoplasmosis previously reported and the 14 here described constitute a substantial body of clinical material from which some general conclusions may be drawn as to the forms of disease with which this infection may be associated in cats.

Acute and Subacute Infections.—Both acute and subacute infections appear to occur in young animals and are characterized by stubborn fevers and unresponsiveness to antibiotics. In the majority, the outstanding symptom is a rapid, labored type of respiration resulting from bronchopneumonia and unaccompanied, apparently, by coughing. In other cats, although dyspnea may develop terminally, the symptoms may be those of an acute systemic infection of the type of panleukopenia or of a severe abdominal disorder, such as hepatitis, pancreatitis, or intestinal obstruction—the latter impression deriving from enlargement of the mesenteric nodes. Substantial effusion into the serous cavities appears to be the exception rather than the rule. Jaundice and terminal hemorrhages suggestive of liver failure may occur. Central nervous symptoms do not seem to be frequent in acute toxoplasmosis, although organisms may be found on histological examination.

The more extensive the organic involvement, the more severe are the symptoms and the more rapid the course of disease. Death may occur within a few days of onset, but when infection affects mainly the lungs, the illness may be less acute and the course more protracted.

Radiographs of dyspneic cats show lung changes similar to those in inhalation pneumonia—numerous irregular areas of density, particularly in the lower parts of the lungs. When there is extensive involvement of abdominal organs, there may be sufficient peritoneal effusion to obscure the picture.

Blood findings, since the infective organism is practically never observed in films, are not pathognomonic. Either moderate leukocytosis or leukopenia may occur, with available evidence suggesting a trend toward the latter as the disease progresses. In the few differential counts done, neutrophils predominated over lymphocytes, even in leukopenic animals, and monocytes were sometimes increased. Platelets were often substantially decreased. Anemia was not conspicuous.

Allowance being made for the fact that necropsy studies did not, in general, include tissues apparently unaffected, the lungs, liver, spleen, and lymph nodes seem to be the organs predominantly involved, undergoing changes of acute and subacute inflammation and necrosis. Grossly, the lungs have an appearance consistent with foreign-body pneumonia. As previously observed in the cat,[12] as well as in other animals and man, microscopic examination discloses a bronchopneumonia notable for

epithelial proliferation, with Toxoplasma bodies occurring in hyperplastic epithelial cells and macrophages. Liver lesions are those of acute and subacute hepatitis, organisms parasitizing both Kupffer and, less frequently, hepatic cells. The spleen may enlarge, and the lymph nodes, especially the mesenteric, may be swollen and reddened, with Toxoplasma bodies, single or aggregated, in macrophages and endothelial cells. Occasionally, the pancreas may be inflamed and necrotic, or the intestine ulcerated. The severe inflammatory reaction to the organisms observable in the lesions of the acute and subacute infection leaves little doubt that there is a direct relationship between the presence of the organism and the clinical signs.

Although central nervous symptoms are not common in acute infections, there may occasionally be microscopic changes characteristic of nonspecific meningo-encephalitis, or Toxoplasma bodies or cysts may be present without inflammatory reaction.

Chronic Infections.—Apparently, chronic infections occur in older animals, develop, to judge from microscopic evidence, over a longer period, and tend toward localization. In several cats, intestinal granuloma with ulceration simulated lymphosarcoma. The signs were weight loss, diarrhea, and anemia, probably related mainly to the presence of mechanical obstruction.

Another site of localization of Toxoplasma bodies is the central nervous system, both brain and spinal cord. One cat so affected showed no signs of illness; in the others, incoordination, ataxia, and paresis were attributed to lesions of meningo-encephalomyelitis. Found in association with some of these lesions were Toxoplasma bodies and "pseudocysts."* The extent to which this organism was responsible for the lesion is not certain, since

*Although organisms may be absent, it is believed that certain types of inflammatory lesions represent sites of earlier rupture of "pseudocysts," followed by release and destruction of organisms.[5]

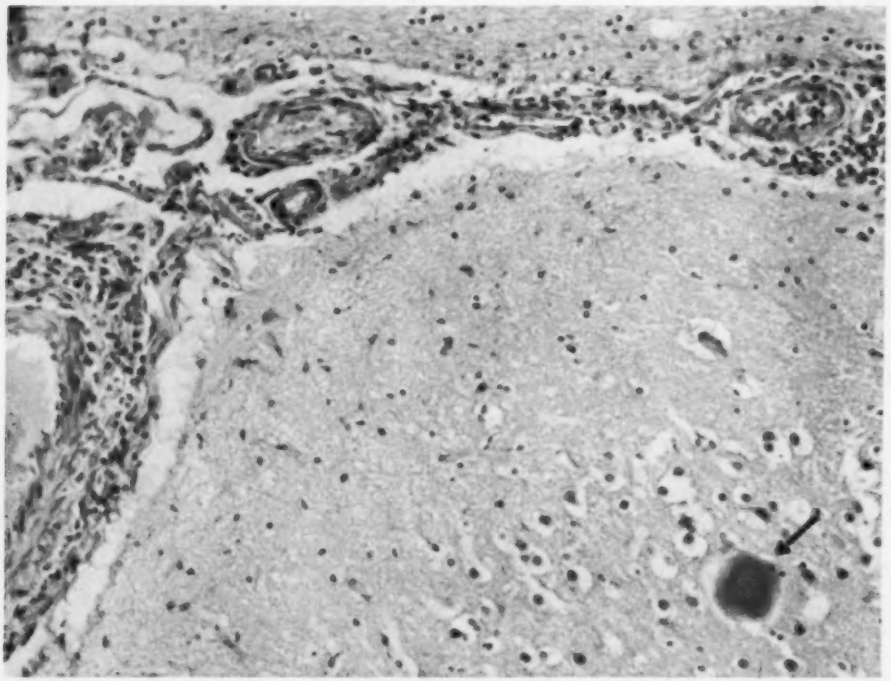

Fig. 10—Brain tissue of a cat, with chronic meningo-encephalitis, associated with presence of Toxoplasma "pseudocysts" (arrow). Near the cyst there is vacuolation but no inflammatory reaction. The leptomeninges (left and upper fields) are infiltrated with round cells, but no Toxoplasma bodies can be seen. H & E stain; x 400.

absence of reaction around the "pseudocysts" themselves is notable.

Why acute infection should predominate in younger animals and localized lesions in older ones is at the moment a matter for speculation. Conceivably, a degree of immunity may develop with age. Two facts might support this hypothesis: In the generalized infections seen in younger animals, organisms are numerous and tend to result in inflammatory changes and necrosis; whereas in the localized lesions of the older cats, organisms are relatively few and stimulate connective tissue proliferation. Secondly, immunological tests, although not yet done extensively, suggest a widespread contact with the infection among cats; approximately a third of the serum samples from cats of two urban areas indicated positive titers for toxoplasmosis.[6,11]

There is also the possibility that lesions in older animals represent residual infection from a much earlier acute episode.

Differential Diagnosis.—For the small animal practitioner, the material surveyed presents fairly characteristic clinical patterns, but a number of conditions far commoner among cats must first be excluded as possibilities, and it would seem worthwhile to review them in some detail in order to forestall injudicious diagnoses of toxoplasmosis.

Respiratory disorders from which pneumonic toxoplasmosis must be differentiated are, notably, infectious pneumonitis (coryza), inhalation pneumonia, pyothorax, and other abnormal conditions of the chest in which lung expansion is limited.

Pneumonitis, while often a stubbornly febrile disease, is usually manifested as an infection of the upper respiratory tract, with conspicuous salivation, sneezing, lacrimation, and ocular discharge. Rarely, it begins with a laryngopharyngitis that simulates choke. In the few cats in which a recognizable bronchopneumonia develops, a previous history of upper respiratory symptoms usually betrays the cause.

Inhalation pneumonia, while perhaps indistinguishable radiographically from toxoplasmosis, may often be differentiated by a history of habitual dosing with mineral oil or administration of other oral medication, a prolonged "downhill" course, and fever that is usually moderate and often intermittent.

In contrast to toxoplasmosis, in which respirations are accelerated and abnormal sounds are usually heard over the whole chest, hemothorax, pneumothorax, diaphragmatic hernia, pyothorax, and effusions associated with tumors and heart and liver disease to a great extent muffle lung sounds and result in slow, labored, abdominal breathing. Lateral radiographs taken with the cat standing, and study of any material aspirated from the thorax, usually establish a diagnosis in these conditions.

When enlargement of the mesenteric nodes is significant, intestinal foreign body, intussusception, strangulation, or tumor may be suspected. Differentiation by radiograph is sometimes possible, but an exploratory operation may be required for certainty. Acute hepatitis and pancreatitis are not often diagnosed clinically in cats, yet necropsies show that neither is rare. Since both may occur in toxoplasmosis, a suspicion of either would by no means eliminate the possibility of Toxoplasma as the etiological agent.

Specific infectious anemia and febrile anemias due to other types of infection or to leukemia should rarely be confused with toxoplasmosis, since profound anemia is not a common characteristic of the latter.

Panleukopenia (infectious enteritis) has long been the refuge of the practitioner confronted with a feverish cat that refuses to eat and vomits. If a leukocyte count is low, the diagnosis is usually considered established, despite the fact that leukopenia may occur in a variety of other conditions, notably infections, poisonings, and leukemia. A thorough history and examination, vigilant observation, and more extensive blood and necropsy studies would probably show that many diagnoses of panleukopenia are unfounded. While it is possible that toxoplasmosis may in some instances simulate panleukopenia, even in respect to the low leukocyte count, development of pneumonia or jaundice or both might well suggest the former. As to the differential leukocyte count, a relative lymphocytosis is associated with panleukopenia, while in toxoplasmosis neutrophils may outnumber lymphocytes, and platelets are decreased.

That it is not impossible to make a correct clinical diagnosis of acute toxoplasmosis is indicated by case 1 in this paper, in which the combination of fever, jaun-

dice, and rapid, labored respirations seemed practically pathognomonic.

Chronic, localized forms of toxoplasmosis, it has been seen, tend to involve either the intestine or the brain. The intestinal granuloma must be differentiated clinically from a foreign body, fecal mass, chronic intussusception, and annular lymphosarcoma. With the latter, even radiographs and the gross appearance of the lesion at operation may not permit differentiation, but a rapid diagnosis is possible by means of Wright- and Giemsa-stained imprints.

Without serological tests or diagnostic procedures involving spinal fluid, it is unlikely that toxoplasmosis of the central nervous system can be differentiated clinically from a host of other disorders—traumatic, infectious, toxic, and neoplastic—capable of producing similar signs. The problem is sometimes further complicated by uncertainty as to whether the Toxoplasma organisms recognized in a brain are the cause of the manifestations and lesions observed. Organisms may even be present without clinical signs or tissue reaction.

Suffice it to plead for careful necropsies and microscopic study of all cats with behavior suggestive of brain or cord lesions. Brain disorders are among the most common and puzzling problems in the cat, and while for practical purposes it may be sufficient to hazard a guess as to the area involved and to tell an owner that most such disorders are progressive and incurable, it is urgent that their etiology be better understood than at present.

While it has been the purpose of this paper to call attention to certain disease patterns as being suggestive of toxoplasmosis, it must be emphasized that only by laboratory diagnosis can a suspicion of toxoplasmosis be confirmed. In the live animal, resort must be had to immunological tests, microscopic examination of infective material, or recovery of the organism by egg or tissue culture, or by inoculation of animals (guinea pigs, hamsters, or preferably mice). Diagnostic tests in use at present are the toxoplasmin intradermal test, the complement-fixation test, and the Sabin-Feldman dye test for cytoplasm-modifying antibody. Usefulness of the skin test is, in general, limited to chronic and recovered cases. An advantage of the dye test is that it may disclose significant positive titers not merely in brief, acute illness but, as in 2 cases reported in this paper, almost from the onset of disease. Disadvantages are that performance of the test involves the use of live organisms and that at present there are few places at which the test is done. In contrast to dye-test antibodies, which develop early and may persist throughout life, complement-fixing antibodies do not reach their peak for a month or more and then disappear within a few years.

If identification of the organism is to be attempted, either by microscopic examination of infective material or by animal inoculation or culture, tissue specimens or inflammatory exudate from the body cavities must be obtained. Blood is not satisfactory for these purposes, since Toxoplasma is rarely seen in blood films, and infection is not commonly transmissible by inoculation of blood. Tissue imprints or smears of fluid obtained from live animals or made at necropsy, and stained with Wright's or Giemsa's stains, may well permit a rapid and fairly reliable diagnosis of toxoplasmosis, although there exists a possibility of confusion with nuclear debris.

While recovery of the organism through animal inoculation or egg or tissue culture is considered to provide the only indisputable proof of infection, it is likely that, except in cases where the clinical or necropsy picture suggests this procedure, most diagnoses of toxoplasmosis will continue to be made only as a result of microscopic study of tissue sections.

Treatment.—Treatment, disappointing in the past, would now seem to have a prospect of success, although it is possible only if a definite diagnosis of toxoplasmosis can be made and specific therapy started early enough to affect the course of illness. A combination of pyrimethamine and sulfonamides, apparently curative in several infections in man, seemed to be associated with recovery in the one nonfatal infection reported in this paper.

Natural transmission of the disease from animal to animal does not appear to occur readily and, apart from laboratory accidents, there are no authenticated cases of infection passing between animals and man. Yet, since the presence of organisms in pulmonary alveoli and intestinal mucosa raises the possibility of the shedding of

Toxoplasma by sick animals, suspects should be isolated and should be handled and treated with proper precautions against scratches, bites, and dissemination of infection through association or contaminated material.*

SUMMARY

Toxoplasmosis in the cat is described on the basis of cases already reported and of 14 additional ones recognized at the Angell Memorial Animal Hospital in the years 1952 to 1956.

Acute toxoplasmosis occurs in young and young adult cats and is characterized by persistent high fever and unresponsiveness to antibiotics. While the disease may sometimes simulate panleukopenia or other acute abdominal disorders, with jaundice present in some instances, the most conspicuous and typical sign is rapid, pneumonic breathing of progressive severity. The characteristic dyspnea, together with jaundice, should be considered highly suggestive. Leukopenia is a frequent, although not pathognomonic, finding; neutrophils may outnumber lymphocytes, monocytes may be increased, and platelets may be diminished.

Chronic forms of toxoplasmosis seen in older cats may affect the intestine or central nervous system. Granulomas of the intestine may be misdiagnosed as tumors, foreign bodies, intussusceptions, or fecal masses. In the central nervous system, the organisms, themselves stimulating no apparent inflammatory reaction, may be associated with, although perhaps not always responsible for, chronic progressive manifestations of incoordination and convulsions.

Injudicious clinical diagnoses should be avoided by careful differentiation from other far more common disorders of cats. Positive diagnosis can only be accomplished by laboratory procedures, The sole indisputable proof of the diagnosis is considered to be provided by recovery of the organism through animal inoculation or egg or tissue culture of infective material. Other diagnostic procedures are the toxoplasmin skin test, the complement-fixation and dye serological tests, and microscopic examination of exudates, tissue imprints, and tissue sections.

Experience with 1 cat suggests that, provided a timely diagnosis can be made, pyrimethamine (Daraprim), used together with sulfonamides, may exert a favorable influence on the course of infection.

*The two persons responsible for all treatment and handling in the 2 known cases of infection reported in this paper observed these precautions and after 4 weeks' association, including an occasional scratch, still had no antibody titer by the dye test. The four members of the family in which the cats were house pets also had negative serums.

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Virus of Foot-and-Mouth Disease Photographed

The virus of foot-and-mouth disease (aftosa) has been purified, isolated, and photographed by scientists at the Plum Island Disease Laboratory. The electron micrographs show the virus to be spherical and about 0.000001 of an inch in diameter, the smallest of the viruses known to affect animals. It is even smaller than human poliomyelitis virus.—*U.S.D.A. Release, Sept. 9, 1957.*

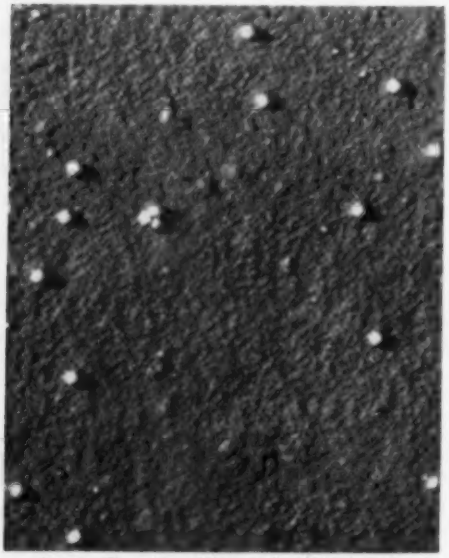

Fig. 1—The virus of foot-and-mouth disease.

Orphan Viruses Found in Animals.—In recent years, a number of viruses called "orphans," because the diseases to which they belong are unknown and they do not produce illness in laboratory animals, have been found in the intestines of man. They are known as the "ECHO" group, which means "enteric cytopathogenic human orphan" virus. Now viruses named ECMO, ECBO, and ECSO have been isolated from the intestines of monkeys, cows, and pigs, respectively. Families of viruses like the herpes and pox viruses have been known to exist both in animals and man but enteric viruses have not previously been found except in man.—*Sci. News Letter (Sept. 21, 1957): 184.*

Antibiotics in Virus Diseases.—The action of antibiotics in disease differs radically from the action of antiserums. Except for the sensitization phenomena, an antiserum which does no good at least does no harm. However, when an antibiotic encounters a resistant organism, its side effects become its only effects. All viruses, except the large particle ones such as those of trachoma and psittacosis, are resistant to the known antibiotics. Virus diseases are systemic infections and, although the patient shows little illness, his defense mechanism must be considered to be under serious strain. At such a time, disturbing the physiological flora of his body with chemotherapeutic drugs and antibiotics could possibly have grave consequences.—*J.Am.M.A. (Sept. 7, 1957): 53.*

Rats Frozen, then Revived.—As the body temperature of experimental rats approaches freezing, breathing decreases and stops then, finally, the heart stops. If the rats have breathed a high concentration of carbon dioxide gas while being cooled, they can be revived up to one hour later with artificial respiration, using a high concentration of oxygen. The heart rhythm returns in ten minutes and a regular breathing in another 20 minutes.—*Sci. News Letter (Sept., 1957): 152.*

The homing ability of bats was shown to be not dependent on vision when those which were blindfolded returned to their cave from 5 miles away as rapidly as did controls. When 484 bats were released at dusk, various distances from their cave, 6 per cent of the 60-mile group and 72 per cent of the 5-mile group returned before morning. Some traveled at average speeds of 10 to 19 miles per hour.—*Science (Aug. 16, 1957): 307.*

Surgery and Obstetrics
and Problems of Breeding

Effect of Preanesthetic Medication with Chlorpromazine, Meperidine, and Promazine on Pentobarbital Anesthesia in the Cat

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THE VALUE of preanesthetic medication has been well established in human anesthesiology.[11] Atropine and morphine have been given to dogs, prior to pentobarbital sodium (Somnnopentyl*) anesthesia for many years.[14] They facilitate restraint and inhibit respiratory secretions and emesis during surgery. They also reduce the required dose of anesthestic, shorten the recovery period, and reduce excitement during recovery. With cats, since morphine may cause excitement, preanesthetic sedation has not been employed routinely.

Recently, it has been demonstrated that meperidine (Demerol†), a drug with activity resembling that of morphine and atropine, is a satisfactory preanesthetic agent for cats.[3,4,7,16] When given intramuscularly (5 mg./lb. of body weight), it was reported to quiet active cats.[3] Following subcutaneous injection of meperidine in low dosage (2.2 to 4.5 mg./lb.), cats were easier to handle but not markedly depressed.[4] When meperidine was given subcutaneously in high dosage (10 mg./lb.), cats were easier to restrain and required less anesthetic.[16] Following the intramuscular (2.5 mg./lb.) or subcutaneous (5 mg./lb.) injection of meperidine, the amount of pentobarbital sodium required to produce surgical anesthesia was reduced from 1 gr. (65 mg.) per 5 lb. of body weight to 1 gr. per 7 lb. or 1 gr. per 6 lb., respectively.[7] The optimum time between injection of meperidine and pentobarbital was approximately one hour for the subcutaneous route and 45 minutes for the intramuscular route. Lack of sedation following the administration of meperidine has been reported.[2,7,8]

Chlorpromazine (Thorazine**), a drug whose chemical formula is similar to that of phenothiazine and promethazine (Phenergan‡) (fig. 1) has been reported to potentiate certain analgesic and anesthetic

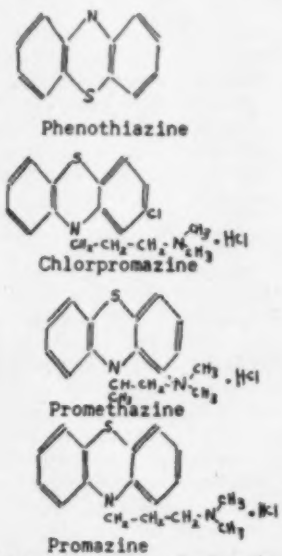

Fig. 1—Structural formulas showing the chemical similarity of four agents.

agents.[15,17,21] Investigators have observed prolongation of sleeping time§ when chlorpromazine was given prior to various barbiturates in dogs, mice, and rats.[1] Several authors have reported favorable results with chlorpromazine and pentobarbital in canine surgery.[6,9,15,20,22]

In cats,[5] depression, ataxia, and relaxation of the nictitating membrane were slight and disappeared after 24 to 48 hours following small (0.7 mg./lb.) doses of chlorpromazine, but were marked and lasted from two to three days following a larger dose (1.1 mg./lb.).

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Paper No. 3781, journal series, Agricultural Experiment Station, University of Minnesota.

*Somnnopentyl, the trade name for pentobarbital sodium, is produced by Pitman-Moore Co., Indianapolis, Ind.

†Demerol, the trade name for meperidine, is manufacturned by Winthrop-Stearns, Inc., New York, N.Y.

**Thorazine (chlorpromazine) is produced by Smith, Kline, and French Laboratories, Philadelphia; distributed by Pitman-Moore Company, Indianapolis, Ind.

‡Phenergan, the trade name for promethazine, is produced by Wyeth Laboratories, Philadelphia, Pa.

§The anesthetic drugs are administered at the same dosage.

TABLE 1—Effect of Pentobarbital on Rectal Temperature and Time of Return of Righting Reflex Following Ovariohysterectomy in the Cat

Age	Breed	Wt. (lb.)	Pentobarbital (gr.)	Pentobarbital (lb./gr.)	Lowest temp. (F.)	Righting reflex (hr.)
6 mo.	D.S.H.*	5.3	1.2	4.4	— — **	5.0
3 yr.	D.S.H.	7.0	1.6	4.3	97	7.0
18 mo.	D.S.H.	7.4	2.4	3.0	97	7.0
Adult	D.S.H.	5.7	1.0	5.7	98	6.0
6 mo.	D.S.H.	4.7	1.7	2.8	— —	5.8
5 mo.	D.S.H.	4.9	0.6	8.2	— —	8.5
6 mo.	D.S.H.	4.3	1.2	3.5	— —	8.0
18 mo.	D.S.H.	5.2	0.8	6.5	— —	10.0
5½ mo.	Siamese	5.0	1.0	5.0	— —	7.5
Adult	D.S.H.	5.4	1.6	3.4	97	12.5
Average	... ...	.....	.....	4.7	97.3	7.7

*Domestic short-haired cat. **Rectal temperatures not taken during recovery.

Promazine (Sparine‖) has the same basic structure as chlorpromazine but lacks a chloride atom on the phenothiazine nucleus (fig. 1). Its actions are similar to those of chlorpromazine. Although it has been used primarily in psychiatry,[10] its clinical use has been reported in the veterinary literature.[12,13]

EXPERIMENTAL PROCEDURE AND RESULTS

In a series of studies with experimental cats and a limited number of clinical patients, it was observed that chlorpromazine, promazine, and meperidine alone produced poor sedation and did not markedly influence the anesthetic dose of pentobarbital, when given subcutaneously. However, combinations of chlorpromazine and meperidine, or promazine and meperidine produced sedation, reduced the anesthetic dose, and shortened the recovery period. The latter effects are important, since pentobarbital has a depressing effect on the respiratory system and cats may die during a long recovery period. The next procedure was to give preanesthetic sedatives, alone or in combination, to all normal cats admitted to the veterinary clinic for ovariohysterectomy. Most of these operations were done on the day of entry. To reduce pain from injection, 1 ml. of 2 per cent procaine solution was added to the sedative solution. It had been observed that, to obtain the same preanesthetic and anesthetic effects, it was necessary to give twice as much promazine as chlorpromazine. Cats given 2 mg. of chlorpromazine per pound exhibited side effects, e.g., ataxia, mewing, extension of the forelegs and claws, and postoperative sedation, whereas these effects were not observed with a similar dose of promazine. The concentration of chlorpromazine and promazine injected subcutaneously was 5 mg. per milliliter. The meperidine solution (50 mg./ml.) was not diluted. Five milligrams of meperidine was injected per pound of body weight.

Approximately one hour after preanesthetic medication, pentobarbital sodium (diluted to 0.5 gr./ml. with sterile distilled water) was injected slowly into the cephalic vein until palpebral, corneal, lingual, jaw, ear-whisker, and digital reflexes were abolished. In the recovery period, pulse rate, rectal temperature, respiratory rate, color of mucous membranes, pupil size, shivering, stretching, righting, and standing were recorded at half-hour intervals (tables 1 to 5). No data on resistance to venipuncture are included.

The cats which were given combined preanesthetic medication showed more depression and were easier to restrain than those given a single agent. The effects of combined preanesthetic medication were

‖Sparine, the trade name for promazine, is produced by Wyeth Laboratories, Philadelphia, Pa.

TABLE 2—Effect of Chlorpromazine and Pentobarbital on Rectal Temperature and Righting Reflex Following Ovariohysterectomy in the Cat

Age	Breed	Wt. (lb.)	Chlorpromazine (mg.)	Pentobarbital (gr.)	Pentobarbital (lb./gr.)	Lowest temp. (F.)	Righting reflex (hr.)
6 mo.	D.S.H.	6.2	6	1.5	4.1	96.6	8.3
5 mo.	D.S.H.	5.0	5	0.8	6.6	99.0	7.0
19 mo.	Siamese	7.1	7	0.7	11.1	—	8.0
5 mo.	D.S.H.	7.0	7	1.5	4.7	100.4	9.5
4 mo.	D.S.H.	5.5	5	1.2	4.6	94.0	7.5
Average	... ...	.....	....	.....	6.2	97.5	8.1

TABLE 3—Effect of Promazine and Pentobarbital on Rectal Temperatures and Righting Reflex Following Ovariohysterectomy in the Cat

Age	Breed	Wt. (lb.)	Promazine (mg.)	Pentobarbital (gr.)	Pentobarbital (lb./gr.)	Lowest temp. (F.)	Righting reflex (hr.)
2 yr.	D.S.H.	8.0	16	1.2	6.7	—	1.3
13 mo.	Siamese	7.5	14	1.5	5.0	99.0	4.0
16 mo.	D.S.H.	7.0	14	1.2	5.8	98.0	4.0
6 mo.	Siamese	5.2	10	1.3	4.2	94.0	7.3
6 mo.	D.S.H.	6.3	12	1.1	5.7	94.3	11.0
Average	.....	...	...	...	5.5	96.3	5.5

usually evident in 15 to 20 minutes and reached a maximum in one hour. Most cats showing sedation crouched quietly with their eyes partially closed, the pupils dilated, and the nictitating membranes showing partially.

Cats given chlorpromazine and meperidine in combination were frequently ataxic. If restrained for several minutes, some would extend their forelegs and claws, cry out, and struggle similarly to those given no preanesthetics or given only one agent. Struggling was not alleviated by higher doses of chlorpromazine. With either combination of drugs, some cats appeared to be aroused during restraint and venipuncture, but this seldom occurred if handled gently and quietly and not over-restrained.

The mean anesthetic dose of pentobarbital in cats without preanesthetic sedation was 1 gr. per 4.7 lb. The preanesthetic combinations of chlorpromazine-meperidine or of promazine-meperidine decreased the dose to 1 gr. per 8.1 lb. and 7.5 lb. of body weight, respectively. The lowest mean rectal temperatures following surgery were 97.3 F. for pentobarbital anesthesia; 96.5 F. for chlorpromazine-meperidine-pentobarbital anesthesia, and 96.9 F. for promazine-meperidine-pentobarbital anesthesia. The mean time intervals between anesthetization and return of the righting reflex

TABLE 4—Effect of Meperidine and Pentobarbital on Rectal Temperature and Righting Reflex Following Ovariohysterectomy in the Cat

Age	Breed	Wt. (lb.)	Meperidine (mg.)	Pentobarbital (gr.)	Pentobarbital (lb./gr.)	Lowest temp. (F.)	Righting reflex (hr.)
4½ mo.	Siamese	4.5	25	0.6	7.5	99.4	6.0
Adult	D.S.H.	5.8	30	1.4	4.1	95.5	7.5
5 mo.	D.S.H.	4.9	25	1.0	4.9	96.0	8.5
8 mo.	D.S.H.	6.4	30	1.3	4.9	96.8	6.5
6 mo.	D.S.H.	5.1	25	1.2	4.3	95.4	6.0
Average	.....	...	...	...	5.1	96.6	6.9

TABLE 5—Effect of Chlorpromazine and Meperidine and Pentobarbital on the Rectal Temperature and Righting Reflex Following Ovariohysterectomy in the Cat

Age	Breed	Wt. (lb.)	Preanesthetic (mg.)	Preanesthetic (agent)	Pentobarbital (gr.)	Pentobarbital (lb./gr.)	Lowest temp. (F.)	Righting reflex (hr.)
Adult	D.S.H.	4.5	5 25	Cpz. Mep.	0.5	9.0	—	3.8
2 yr.	D.S.H.	9.7	10 50	Cpz. Mep.	1.0	9.7	—	8.0
1 yr.	D.S.H.	6.0	6 30	Cpz. Mep.	0.5	12.0	—	5.5
6 mo.	D.S.H.	4.4	3 25	Cpz. Mep.	0.8	5.8	93.6	1.0
5 mo.	Siamese	4.5	5 25	Cpz. Mep.	0.8	6.0	98.0	6.0
9 mo.	Persian	10.0	10 50	Cpz. Mep.	1.5	6.7	99.5	4.0
4½ mo.	D.S.H.	4.5	5 25	Cpz. Mep.	0.6	7.5	—	2.5
4 mo.	Siamese	5.0	3 25	Cpz. Mep.	0.5	10.0	93.8	7.0
6 mo.	D.S.H.	6.4	6 30	Cpz. Mep.	0.9	7.0	96.0	9.0
6 mo.	D.S.H.	6.0	6 30	Cpz. Mep.	0.8	7.5	95.6	5.5
Average	.....	...	...	.....	...	8.1	96.5	5.2

TABLE 6—Effect of Promazine, Meperidine, and Pentobarbital on Rectal Temperature and Righting Reflex Following Ovariohysterectomy in the Cat

Age	Breed	Wt. (lb.)	Preanesthetic (mg.)	Preanesthetic (agent)	Pentobarbital (gr.)	Pentobarbital (lb./gr.)	Lowest temp. (F.)	Righting reflex (hr.)
1 yr.	D.S.H.	9.1	18	Prom.	1.2	7.5	95.4	4.0
			45	Mep.				
1 yr.	D.S.H	7.2	14	Prom.	0.8	9.6	—	3.0
			35	Mep.				
6 mo.	D.S.H.	8.0	16	Prom.	1.0	8.0	98.0	1.5
			40	Mep.				
1 yr.	D.S.H.	9.3	18	Prom.	1.2	7.8	95.4	3.3
			45	Mep.				
Adult	D.S.H.	4.3	8	Prom.	0.8	5.7	95.6	4.5
			20	Mep.				
2½ yr.	Angora	7.0	14	Prom.	1.0	7.0	99.4	2.0
			35	Mep.				
5 mo.	D.S.H.	6.0	12	Prom.	0.5	12.0	—	3.5
			30	Mep.				
1½ yr.	D.S.H.	7.9	16	Prom.	2.0	4.0	98.0	5.0
			40	Mep.				
4 mo.	D.S.H.	4.3	8	Prom.	0.7	6.1	94.6	4.5
			20	Mep.				
2 yr.	D.S.H.	5.3	10	Prom.	0.8	7.0	99.0	6.0
			25	Mep.				
Average	.....	....	....	.....	....	7.5	96.9	3.7

were 7.7 hours for pentobarbital anesthesia; 5.2 hours for chlorpromazine-meperidine-pentobarbital anesthesia, and 3.7 hours for promazine-meperidine-pentobarbital anesthesia.

When submitted to the "t" test,[10] the reduction in the required dose of pentobarbital with both combinations of preanesthetic agents was significant at the 1 per cent level, and the reduction in time required for righting was significant at the 5 per cent level for chlorpromazine and meperidine and at the 1 per cent level for promazine and meperidine.

Some cats given combined preanesthetic medication regained their other reflexes more rapidly than their ability to right themselves. The duration of surgical anesthesia was estimated to vary from one half to one hour in cats showing preanesthetic sedation and from one to three hours in those not showing it.

Both combinations of preanesthetic agents make pentobarbital anesthesia safer by reducing the dose of pentobarbital, by facilitating intravenous administration, and by shortening the recovery period as measured by the return of the righting reflex. There was also less emergence excitement during recovery. Additional studies are needed to determine if there is a greater margin of safety during surgical anesthesia.

SUMMARY

Cats which were given chlorpromazine, promazine, and meperidine alone and in combination prior to pentobarbital anesthesia were compared to a control group which was anesthetized with pentobarbital without preanesthetic sedation. Both combinations of sedative agents significantly reduced the dose of pentobarbital required for surgical anesthesia and significantly hastened the return of the righting reflex. Cats given preanesthetic medications were easier to restrain for venipuncture.

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Puerperal Tetany in the Bitch.—This condition (eclampsia) is difficult to treat intravenously because of the twitching and because the rapid influx of calcium may cause cardiac disturbances. All of 30 cases were successfully treated by the subcutaneous injection of calcium gluconate solution to which was added 3 mg. of hyaluronidase per 20 ml. Marked improvement usually occurred in 15 to 20 minutes, but if the animal was not then walking normally, more was injected. Two animals had relapses but responded quickly to further injections. It is recommended that the pups be removed for 12 hours, that supplementary feeding be initiated, and that the bitch be given a preparation of calcium, phosphorus, and vitamin D for ten days.—*R. W. Gee in Austral. Vet. J. (June, 1957): 152.*

Prolapse of the Uterus in a Mare.—A Thoroughbred mare which had foaled an hour previously was found unable to rise, with a temperature of 98 F., pulse fast and weak, and with her uterus prolapsed, slightly lacerated and bleeding, but with the fetal membrane still attached. When given 9 ml. of 2 per cent procaine epidurally, she arose. The membranes were then removed and the uterus was cleaned and replaced with little difficulty. She was given antibiotics in the uterus and intramuscularly, plus injections of pituitrin (5 ml.) and tetanus antitoxin. Her condition deteriorated rapidly and soon she was unable to lift her head. She was then given 1,000 ml. of 10 per cent dextrose intravenously and a transfusion of 2.5 liters of blood. An hour later, she was on her feet and made a splendid recovery. There was no vaginal discharge and estrus occurred eight days later.—*A. B. Maclean in Austral. Vet. J. (June, 1957): 149.*

An Improvised Thomas Splint for a Bull

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We recently were so pleased with the results from an improvised aluminum Thomas splint applied to the fractured leg of a young bull that it seems worthwhile to describe it. The advantages of this splint are: light weight, easily accessible materials, adequate strength, and reasonable cost.

On June 28, 1956, a client some 200 miles distant phoned to report that a prized young Charolais bull had suffered a broken left hindleg. Newly acquired, he had accidentally gained access to a pen in which other bulls were confined and had incurred the injury during the inevitable fight. After promising to examine and treat the bull on the following morning, the problem of splint materials had to be dealt with immediately. The materials usually used for bovine fracture fixation seemed either undesirable or too complicated for these circumstances. A number of items of "do-it-yourself" aluminum were purchased at a local hardware store, including several 6-ft. lengths of ¾-inch hollow rods, some flanged butt connectors drilled for screws, and some "T" connectors.

Examination of the patient, a 900-lb. bull about 14 months old, confirmed the owner's diagnosis and placed the fracture site in the distal third of the left femur.

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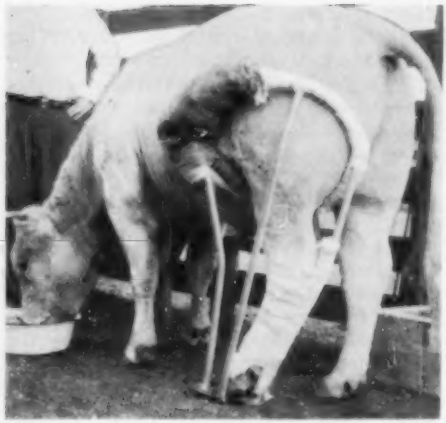

Fig. 1—Young Charolais bull with fractured femur wearing modified Thomas splint made of aluminum.

The limb was slightly swollen above the stifle joint only and crepitation at the fracture site was readily detected.

A modified Thomas splint (fig. 1) was fashioned from the aluminum pieces in the following manner: one rod was packed with wet sand and bent into a ring. The customary two vertical pieces were fastened to this ring, by means of "T" connectors, which were also packed with sand to permit bending slightly to conform to the angulation of the limb. (After completion of the bending, the sand was removed.) A welder was employed to make a bottom piece (*see* figure 1) from boiler plate, using a cardboard pattern which allowed not only space on which the entire foot would rest, but also a lip anterior and posterior to which butt connectors could be bolted for joining it with the vertical pieces. Finally, a third vertical rod, from the ring to the foot piece was added laterally for extra strength. The ring was fitted snugly medial to the thigh to act as a crutch. To hold the splint in place, the foot was wired to the foot plate by means of holes drilled through the hoof walls and the plate.

Examination and application of the splint were accomplished without sedation, thanks to the gentle nature of the patient and the assistance of an attendant. Because of distances involved, only one visit to the ranch was warranted, but the bull walked with the splint immediately and could recline and arise with only slight difficulty. Instructions were left regarding minor adjustments to the padding on the splint and to the duration of fixation (1 mo.). Word was later received that the splint had remained in place for a month, but we have had no subsequent opportunity to examine the patient.

A Hereditary Lameness in Cattle

A condition, recognized in Germany for a quarter of a century as "contraction of the Achilles tendon," has appeared in Britain in the offspring of Fresian cattle imported from Germany because of their straight hocks, with the intent to improve the conformation of British Fresian cattle. The lameness usually appears in calves 6 weeks to 6 months old. However, prior to this, the hindlegs can be noticed to be abnormally straight, with the os calcis often drawn toward the tibia and the hind digits more vertical than normal. The condition usually develops faster in one leg and, in walking, the foot of that limb occasionally misses the ground, with the leg extending stiffly backward, the stifle slightly flexed, and the Achilles tendon spastic. In a few weeks, this leg appears to be shortened, adducted, and may swing like a pendulum when the calf walks. The fetlock of the other hindleg may sink to the ground when bearing weight.

The following operation, if performed as soon as the condition is detected, is usually successful. Under local infiltration anesthesia, with the calf standing, the gastrocnemius tendon is completely divided transversely; then, at the same level, the superficial flexor tendon is partially divided until the foot rests on the ground. The leg is then usually normal within a month. Recovered animals should not be used for breeding; 1 such bull sired several calves with this defect.—*C. Formston and E. W. Jones in Brown Swiss Bull. (Oct., 1957): 11.*

Bovine Viral Vaginitis.—A filterable virus was isolated from two dairy herds with breeding problems characterized by a purulent cervicovaginal mucus. A typical vaginitis occurred in 2 yearling heifers 48 hours after inoculation of the virus into the vagina, and in 4 other heifers after similar inoculation with unfiltered material from infected animals.—*New Zealand Vet. J. (June, 1957): 70.*

Feedlot Tests of the Efficacy of Dow ET-57* (Trolene) for Control of Cattle Grubs

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THE CATTLE GRUBS, or ox warbles, *Hypoderma lineatum* (De Villiers) and *Hypoderma bovis* (De Geer), have been troublesome in the United States for over half a century. Estimates of the annual losses to the livestock industry caused by these pests have ranged as high as $300 million. The Agricultural Research Service, in 1954, reported a $100 million annual loss.

The control of cattle grubs for the past 35 years has depended on applications of rotenone-bearing sprays or dusts at the time when the grubs were encysted in the subcutis of the back of the animal. Rotenone has been reported as killing 48 per cent[3] to 90 per cent[10] of the grubs, with the average being 85 per cent. This is not sufficiently high to eradicate this pest with its reproductive capacity of 600 to 800 eggs per adult female fly.[5]

Attempts at eliminating cattle grubs over an area through the use of rotenone dusts and sprays have been made in a number of European countries, in Canada, and in the United States in South Dakota, Washington, and New Mexico. These have met with fair success, as long as governmental agencies carried out the operation. However, when the governmental agency dropped the tests, farmers were reluctant to carry out the treatments because they were not convinced that grub control was profitable.

The life cycle of the cattle grub is such that difficulty is encountered in bringing the control chemicals and the insect together. The adults of the cattle grub are flies which live for only about one week.[2] These flies follow cattle during the summer months in order to lay eggs on the hair of the legs, flanks, tail, or even on the back. These eggs hatch in two to six days. The young grubs then migrate down the hair and tunnel through the skin at the point where the eggs were deposited. They then spend about five months migrating throughout the animal's body in the connective tissue. The larvae of *H. lineatum* congregate for one or two months in the tissues of the esophagus. Larvae of *H. bovis* are reported to spend a similar period in the spinal canal.[4] They then migrate to the loin region where they become encysted in the subdermal tissue, remaining there for approximately 50 to 60 days. In the cyst on the back, they obtain oxygen through a small hole in the skin. When they have reached full growth, the grubs erupt through these openings and drop to the ground where they pupate under straw, manure, or other protective covering. They remain in the pupal stage until spring; then, when warm weather comes, the adult flies emerge.

With past control practices, the cattle grubs were vulnerable through their life cycle only in two stages: (1) while encysted in the subdermal tissues, where chemicals could be applied to them, and (2) in the adult stage. However, attempts to kill or repel the adult flies have been only partially successful.[8]

With eradication apparently impossible with present control procedures, researchers began turning their attention to possible methods of killing the grubs while they were in the body of the animal. In 1948, a method of transplantation of Hypoderma larvae to rabbits, goats, and mice was used during the testing of approximately 200 chemicals as chemotherapeutic agents.[1] In 1954 and 1955, experiments with systemic insecticides for destruction of cattle grubs were reported.[6,7,9]

In 1955, screening tests at Kerrville, Texas (Laboratory of the Entomological Research Service), revealed a systemic insecticide, an organic phosphate which seemed to be particularly effective in cattle grub control. This material, designated as Dow ET-57* (organic phosphorus compound 0,0-dimethyl-0-2,4,5-trichlorophenyl phosphorothioate) was nearly 100 per cent effective in controlling the grubs when it was given at a dosage of 100 mg. per kilogram of body weight as a capsule, a bolus, or a drench. Toxicity tests indicated that

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*Dow ET-57, to be sold as Trolene, is produced by the Dow Chemical Co., Midland, Mich. In the fall of 1956, capsules of this drug were obtained for testing purposes.

TABLE 1—Number of Grubs in Cattle Treated with Dow ET-57

	Total No. grubs				
	Palpation		At slaughter, April 12, 1957		
	Feb. 15, 1957	March 21, 1957	Carcass	Hide	Accumulative
Treated Dec. 28, 1956	0	0	44	5	49
Untreated	147	200	194	370	911

at this level there were no signs of poisoning apparent in treated animals. This treatment preceded the normal appearance of *H. lineatum* larvae by two to three months. Grubs in treated animals apparently were killed before they reached the animal's back.

EXPERIMENTAL PROCEDURE

This paper reports the results of a test of the efficacy of this agent in destroying the parasite in feeding cattle.

On Dec. 28, 1956, the test was started, using 154 head of Montana and Colorado Hereford steers on full feed.† These animals were weighed and placed in two pens, with one group being designated, at random, for treatment. The treated steers averaged 883 lb., the untreated averaged 903 lb. The 77 treated animals were each given gelatin capsules containing the organic phosphate at a dosage rate of 110 mg. per kilogram of body weight. At the end of the 106-day observation period, each animal was again weighed and then slaughtered.

Twice during the 106-day observation period, 49 and 84 days after treatment, grub counts were made by palpating on the backs of the steers in each group.

Since grubs remain in the subcutis an average of about 50 days and only the large warbles were counted, few if any of the grubs were counted twice. At slaughter, counts were made of grubs still remaining on the "hides" and in the carcasses.

RESULTS

All grub counts are shown (table 1).

Results of these tests indicated a 94.7 per cent reduction of the grubs before they reached the subcutis of the back region. The treated steers each gained an average of 19 lb. more than the untreated animals. The savings in feed alone was $3.88 per head—a total of $298.76 (table 2).

Initially, the treated animals, all of which were on full feed, showed a rather severe intoxication from the treatment. All 77 went off feed for two or three days and did not return to the consumption level of the untreated steers for nearly two weeks. Three of the treated steers showed extreme weakness and inappetence but recovered without treatment.

DISCUSSION

Unpublished reports from Wisconsin, Montana, North and South Dakota, Nebraska, Kansas, and Oklahoma indicate that signs of intoxication may be evident in cattle on full feed and in those not drinking sufficient water. The toxic effects were reproducible in animals that were on full feed and in cold weather when their water intake was low. It appears that this intoxication could be averted by treating animals before they are placed on full feed or withholding water for 24 hours prior to treatment, then allowing them to drink all they desire.

SUMMARY

Recently, 77 steers averaging 883 lb. were treated with capsules of an organic phosphate, Dow ET-57 (Trolene), at the rate of 110 mg. per kilogram of body weight. The animals were observed for 106 days. Grub counts indicated a total of 49 in the 77 treated, and 911 in the 77 untreated steers. Treated animals gained 2.48 lb. per day compared with 2.33 lb. per day

†Cattle and facilities for this test were furnished by Rath Packing Co., Waterloo, Iowa.

TABLE 2—Weight Gains, Feed Efficiency, and Carcass Grades of Cattle Treated with Dow ET-57

	Average gain (lb.)		Lb. of feed per 100 lb. gain	Cost per 100 lb. gain	Carcass grade at slaughter, April 12, 1957		
	Total	Daily			High choice	Low choice	Common
Cattle treated (77) Dec. 28, 1956	264	2.48	979	$27.15	68.75%	31.43%	———
Cattle untreated (77)	245	2.33	1,033	$28.62	71.71%	27.27%	1.02%

for the untreated animals. There was essentially no difference in carcass grades at slaughter. The total saving in feed alone was $3.88 per head, or $298.76 for the 77 treated steers.

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Hypoderma bovis (warble) infection in a calf 8 months old, that had never been at pasture or exposed to the fly, was considered to have occurred *in utero* through the placenta.—*Vet. Bull. (July, 1957): Item 2114.*

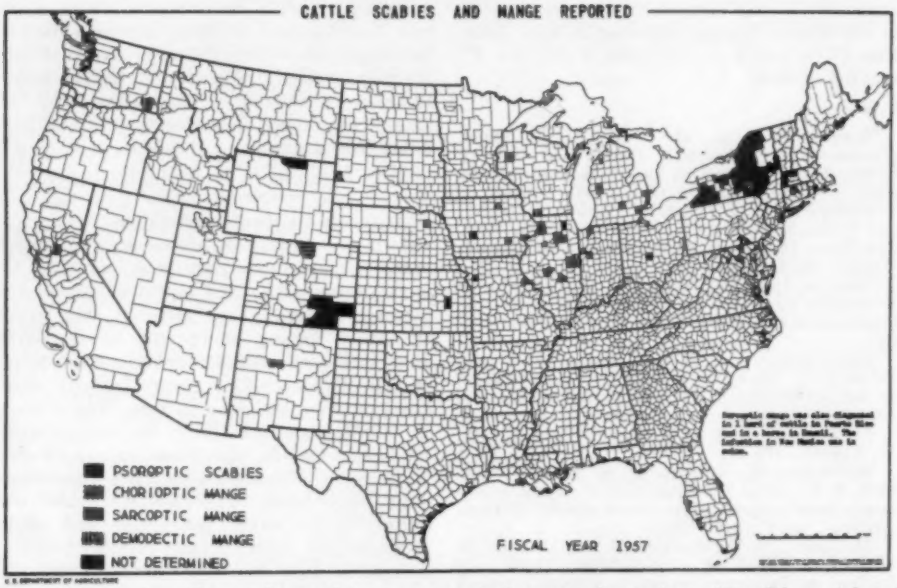
Cryotherapy for Snake Bite in Man.—The commonly recommended tourniquet, multiple incision, and suction does little to alleviate tissue destruction due to the enzyme-like substances in snake venom. Cryotherapy (freezing) prevents rapid absorption of the neurotoxic factor, inhibits bacterial activity, provides effective anesthetic relief, and allows time for activity of antitoxin. If possible, a hemostat should be applied as a ligature just proximal to the bite and the affected part should be immediately immersed in iced water. After not less than five minutes, the hemostat should be removed, and after two hours a pack of finely crushed ice can be substituted for the ice water. Cryotherapy should be continued for 24 hours to three or more days, depending upon the size of the snake. The warm-up of the part must be gradual.—*J.Am.M.A. (Aug. 24, 1957): 1974.*

Anthrax in India.—Anthrax is one of the major livestock diseases in Madras state. Under field conditions, when it occurs among cattle and buffaloes, sheep and goats are usually not affected; and when it occurs among sheep and goats, cattle and buffaloes usually remain unaffected. Rarely, it occurs in a severe form in all of these species and also in man. The disease is usually seen in cattle in the wet, cultivated areas and in sheep and goats in dry, hilly areas. When animals were experimentally inoculated, the bovine strain was found to be lethal for sheep and goats, whereas the ovine strain produced only a subacute form of the disease and the caprine strain produced no disease in cattle. The fact that sheep and goats are seldom affected, when anthrax occurs in cattle, may be due to other factors such as the more prompt and sanitary disposal of cattle carcasses.—*Indian Vet. J. (July, 1957): 258.*

Sulfisomidine, a New Sulfonamide.—Sulfisomidine (Elkosin, Ciba Pharmaceutical Products Inc.), a structural isomer of sulfamethazine, is useful in the treatment of systemic and urinary tract infections. In contrast to most other sulfonamides, it is not readily acetylated; therefore, about 90 per cent appears in the urine in the free form. Since it is soluble in acid urine, renal injury with hematuria and crystalluria is rarely encountered. However, high fluid intake should be maintained.—*Council on Drugs in J.Am.M.A. (Aug. 31, 1957): 2048.*

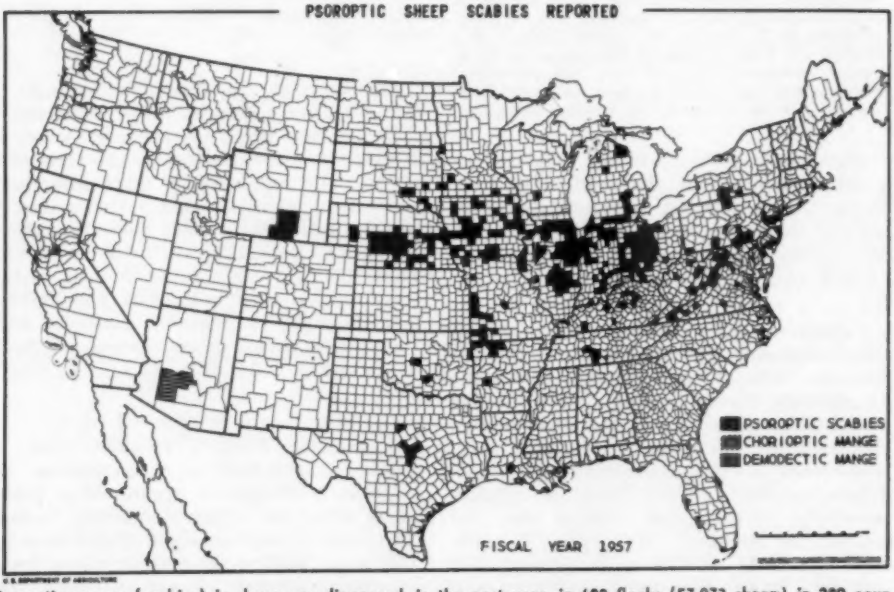
Erysipelas Bacillus Isolated from an Eagle.—Erysipelothrix rhusiopathiae was isolated from several organs of a golden eagle killed in northern Alberta, Canada. The eagle showed no signs of infection but had been feeding on carrion where swine erysipelas existed.—*Canad. J. Comp. Med. (Aug., 1957): 290.*

Scabies in Cattle and Sheep



Psoroptic mange in cattle was diagnosed in seven states in the year ending in June, 1957. All known infected herds have been treated and quarantine areas have been reduced.

Chorioptic mange was found in cattle in 13 states. Sarcoptic mange was found in cattle in three states, in swine in New Mexico, and a horse in Hawaii.



Psoroptic mange (scabies) in sheep was diagnosed, in the past year, in 682 flocks (57,073 sheep) in 289 counties in 24 states; an increase of 75 flocks and 22 counties but in one fewer state than the previous year.

Brisket Disease Syndrome

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Laramie, Wyoming

THE TERM, "brisket disease syndrome," is preferable to "brisket disease" or "altitude disease" because it would appear that altitude is not the only etiological factor. Excellent descriptions of the condition[3,18] have recently become available[7,20] and the changes attributable to altitude have been discussed.[19]

CLINICAL CONSIDERATIONS

A brief description of a typical case will illustrate the salient features of brisket disease syndrome. An Aberdeen Angus, 12 years old, which had apparently been well two days earlier, developed a swollen brisket and two days later was brought to the laboratory. Her condition became worse and, five days after the disease was recognized, she was killed.

On necropsy, the ventral abdominal and perineal regions, flanks, ventral and lateral cervical and thoracic regions, the brachial nerves, and the regional lymph nodes were all edematous. A large amount of blood-tinged fluid was in the thoracic cavity, and the lungs were edematous with congestion along the ventral borders. The heart was normal in size but thin-walled with slight nodular thickening of the atrioventricular valves. Ascites was present. The liver was small, with rounded margins and fibrosis. Sectioning revealed stellate patterns of connective tissue with several small islands of connective tissue and small abscesses in the dark liver tissue. The gallbladder was thick-walled; the bile, viscid. The spleen was small, thin, and dense. The mesenteric lymph nodes were edematous.

Of 56* cattle with brisket disease syndrome available to the Wyoming State Veterinary Laboratory for study over a period of eight years, 35 were Herefords, 18 Aberdeen Angus, 2 Brahman, and 1 a Holstein-Friesian; a higher proportion were Aberdeen Angus as compared with the general cattle population. Of 57 cases, 37 occurred in females, 13 in steers, and 7 in bulls. Nine cases were presented during January, 6 in February, 9 in April, 2 in August, 8 in September, 13 in October, 3 in November, and 6 in December. The higher fall and winter incidence, although it compares closely with that found by others,[18] may not be a true picture since most were range cattle which frequently are not under close observation during the summer. The ages for 56 of the animals were: 16, mature or old; 13, from 1 to 5 years; and 27 were 1 year old or less. This would indicate a high incidence in young animals and another increase in older ones.

Information indicating the time necessary for clinical signs to appear was available for 27 animals: 11 animals developed clinical signs in less than one week, 4 in one to two weeks, 7 in two to four weeks, 3 in one to two months, and 2 in over two months. In 2, symptoms became pronounced in two and three days, respectively. Of 54 animals, 36 died (6 were dead when received; 16 lived one to seven days; 5 lived eight to 14 days; 7 lived 15 to 30 days; and 2 lived 31 to 60 days), 11 were killed *in extremis*, and 7 recovered after 14, 14, 19, 21, 28, 28, and 42 days of illness, respectively. Conditions that might have had a predisposing influence were: pneumonia in 3, scours in 1, unthriftiness for two months in 2, and the eyelids of 1 had become swollen after it had been sprayed with DDT suspension a week previously.

The majority of these cases were from the Laramie, Saratoga, and Cheyenne areas where the altitudes are 7,165, 6,786, and 6,062 ft., respectively.

In all cases, a ventral cervical edema, involving the subcutaneous and deeper tissues, extended posteriorly to a variable degree. The animals' appearance indicated ascites, and marked diarrhea was usually present. In all cases, the jugular veins were distended and a distinct jugular pulse was observed. On exertion, respiratory distress

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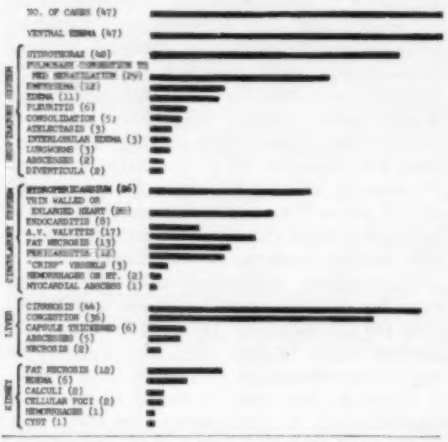
The author thanks the Wyoming practitioners for their cooperation in making these cases available; also the veterinarians at the Wyoming State Veterinary Laboratory for their help; Mr. O. A. Beath, professor emeritus in research chemistry, University of Wyoming, for the selenium analyses; and members of the Armed Forces Institute of Pathology for examining tissues from some of these cases.

*The total number cited for each condition varies, as complete information was not available for all cases.

was readily evident. Some animals remained alert even when unable to rise; others succumbed from the excitement and stress incidental to loading and transport. These were invariably advanced cases, yet symptomatic treatment produced temporary benefit in some and recovery in others.

GROSS PATHOLOGY

Gross changes are summarized for 47 cases (graph 1). The hydrothorax, which



Graph 1—Frequency of gross pathological changes observed in brisket disease syndrome.

was common (85.1%) and frequently pronounced, readily explains the respiratory distress, especially when coupled with excessive ascites. In the lungs, edema, congestion, and changes progressing to red hepatization, usually involving the pendant parts, when combined with the pressure of the increased pleural fluid, could readily result in a compensatory emphysema. In a few instances, the lungs appeared collapsed, probably from excessive pressure from pleural transudate. Endocarditis, especially of the atrioventricular valves, was seen in many, but the valvular lesions, while readily seen and felt, were not of "vegetative" proportions. Whitish plaques were seen on the intimal surface of the larger arteries of 6.3 per cent, and in such cases the normal elasticity of the vessels was impaired. Practically all of the 47 animals had hepatic congestion and cirrhosis, usually with a typical "nutmeg liver" appearance.

In the tabular summary, changes observed in the gastrointestinal tract have been omitted. While 34 had large collections of ascitic fluid, it was completely absent in only a few and probably had been modified by therapy. In about half of these cases, there was gross evidence of edema and necrosis of serosal fat along the intestinal tract.

MICROSCOPIC PATHOLOGY

In explanation of the liver changes (graph 2), several pathological changes might be seen within a single specimen or even in a small area of liver. Recognition of central congestion of the liver lobule was most frequent; this might appear centrally or extend over a large part of the lobule and merge with similar areas in other lobules or even appear as widespread hemorrhage. Next most frequently seen was thickening of the liver capsule. Central fibroblastic tissue and a more dense type of connective tissue increase could be distinguished in 50 per cent. A similar central congestion of the lobule, but ringed with a layer of variable thickness of fatty infiltration of the surrounding parenchymal cells, was seen in 46 per cent. There was central fatty infiltration of parenchymal cells (in 26%), central fibrosis surrounded by fatty infiltration (in 24%), central necrosis (in 14%), central necrosis surrounded by fatty infiltration (in 10%), necrotic foci in which a definite area could not be assigned (in 10%), fibrosis involving the periportal area (in 36%), proliferation of the bile ducts (in 14%), widespread fibrosis (in 8%), and calcification at varied points (in 4%).

Portions of the gastrointestinal tract were available from 48 animals but are not represented in graph 2. Edema, primarily submucosal, (in 56%), fat necrosis in the serosal layer (in 23%), desquamation of surface epithelium (in 12%), and miscellaneous changes such as mucosal cellular proliferation or necrosis, surface ulceration or scarring, and congestion (in 9%) were seen.

The endocrine glands were not routinely examined microscopically in all cases, but necrosis of the pancreatic fat was seen in 11; edema of the pancreatic stroma in 8; edema involved the adrenals in 8; and in 6 animals, the adrenals were normal. Necrosis of fat adjacent to the thyroid gland occurred in 3, and adjacent to the adrenals in 1 animal. Edema was present in one thyroid gland, central hyperemia in one adrenal gland, hemorrhages occurred in one adrenal and one pancreas, and degeneration of the pancreatic islands of Langerhans was detected once.

Of eight brains examined microscopically, edema was present in two, hemorrhage in two, and abscess formation in one.

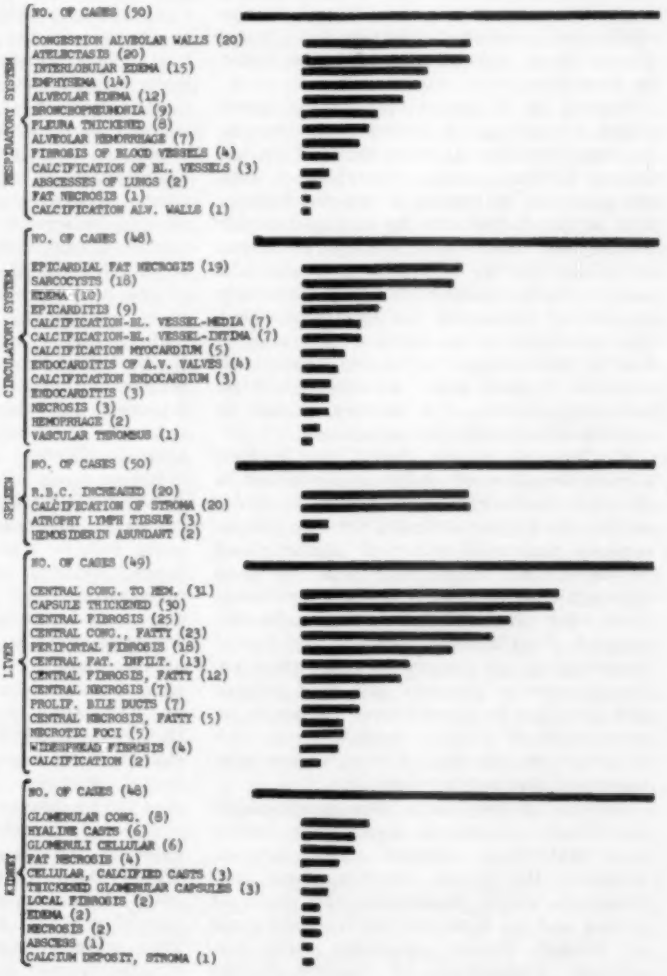
CLINICAL AND LABORATORY TESTS

Using an arbitrary standard of 8 million r.b.c. per cubic millimeter as the approximate normal for cattle at this altitude,[2] the average count for

27 affected animals on which multiple counts were made was below that level in 10, above in 9, and near normal in 8. Where sufficient counts were made in recovered animals to utilize this information, both the hematocrit values and r.b.c. counts increased in 1 case, both remained the same in 1, the r.b.c. count remained the same but the hematocrit values receded in 1, the hematocrit values were unchanged but the r.b.c. count increased in 1, and in 2 animals that recovered both values decreased. Of the cases with fatal termination, in 2 the same approximate erythrocyte count was maintained but hematocrit values were not determined; in 2, the r.b.c. count remained stable but the hematocrit value went down in 1 and up in the other; and in 1 animal, the r.b.c. count decreased but hematocrit values remained essentially unchanged. These values were usually elevated before death so they are not included. Hemoglobin values were usually normal or high (14 to 16 Gm./100 ml. of blood).

Where adequate temperature readings were secured, 4 animals were consistently below 100 F., 13 ranged from 100 to 103, and 12 occasionally fluctuated above 103. Great variations occurred in the leukocyte counts. They were usually high; the high counts coinciding with temperature elevations were believed to indicate secondary infections.

Selenium determinations were made on organs from 9 animals. Only 1 animal had levels of less than 1 p.p.m. of selenium in the liver and kidneys; the highest levels were 2.4, 2.5, and 2.6 p.p.m. in the kidneys of 3 animals. Plasma carotene levels were satisfactory in 4 animals and low in 3; plas-



Graph 2—Frequency of histopathological changes observed in brisket disease syndrome.

ma vitamin A levels were moderate in 4 and low in 3. Serum calcium and phosphorus levels were satisfactory in 3 animals; calcium was high and phosphorus was at a moderate level in 2 others. Lungworms (*Dictyocaulus filaria*) were found in the lungs of 3 animals. The complement-fixation test for anaplasmosis applied to 6 consecutive cases was negative in 2, anticomplementary in 1, and positive in 3.

DISCUSSION

The clinical signs and gross lesions associated with brisket disease syndrome are fairly uniform. While earlier reports emphasized altitude as the causative factor,[7,20] later reports[3,18] tend to discredit this. Undoubtedly, altitude is a factor but generally an accessory one. However, the effects of excessive altitude or abrupt change from low to high altitudes must be considered.[18,25]

Viewed in a broad way, these cases might be considered examples of congestive heart failure. As such, decreased blood volume as from anemia; interference with the elasticity of the blood vessels; restriction of blood flow due to damaged major organs such as the liver, kidneys, or lungs; or failure of the heart as a mechanical pump would possibly be physiologically capable of producing the syndrome. That this condition is not more widely seen is due to the absence of stress from high altitudes in most areas. At extremely high altitudes, anoxia or a relative anemia is capable of initiating this syndrome.

Erythrocyte counts should also include a consideration of their size,[6] although elevated erythrocyte counts have been described for higher altitudes.[14,26] The lymphocytosis associated with high altitudes and increased solar radiation[23] was not seen although, because of complicating infections, the neutrophils were usually increased. Anoxia could increase endothelial permeability to plasma colloids, thus reducing osmotic pressure and fluid balance and leading to circulatory failure,[15] or adrenocortical failure could develop with deranged metabolism of electrolytes with increased capillary permeability.[19]

In none of these cases were exceptionally low blood counts or hematocrit values seen that would suggest anemia *per se*. However, the anemia resulting from anaplasmosis might inaugurate the chain of events and yet disappear by the time signs of brisket disease syndrome were recognized. Comparison of changes in the blood during the disease and after recovery fail to indicate if anemia might have originally been present. In 2 cases, a decrease in hematocrit and erythrocyte levels indicated a return of fluids to the circulation and a reduction in hemoconcentration; in 1, both values remained essentially the same; and in 2, one value was unchanged but the other value changed, indicating an adjustment in the size of the erythrocytes. In the 1 cow, where both values increased with recovery, the elevated leukocyte count also decreased. This indicated that infection, as much as anemia, would have to be considered as a primary cause of this condition.

As stated previously, anoxia might lead to changes in the capillary bed. Calcification (6.3% grossly; 14.0% microscopically) would also have a deliterious effect. Calcification of elements of the vascular system, diaphragm, and spleen have been attributed to a magnesium-deficient diet.[16] Calcification of all layers of the arteries; edema, degeneration, and calcification of skeletal muscle; cloudy swelling and periportal fibrosis of the liver; edema of cardiac muscle; and thickening and calcification of the alveolar walls in animals with low blood phosphorus, high calcium, and normal magnesium have been reported.[1] While these lesions were similar to those due to magnesium deficiency, it would be a conditioned deficiency caused by excessive calcium. Magnesium deficiency has also been implicated in calcareous deposits.[21]

Large doses of calciferol, recommended for the prevention of parturient paresis, produced calcification of the heart and aorta and, in 1 animal, calcification of the lungs, kidney, pleura, peritoneum, and pulmonary vein.[24] Large doses of viosterol may also cause pathological calcification.[4] Areas of necrosis, calcification, and fibrosis of the aorta, at points of maximum pressure, developed in guinea pigs and rabbits on a vitamin E-deficient diet.[12] Intimal thickening, calcification, and hyaline change have been attributed to increased portal pressure from cirrhosis.[27] Veins also can be involved, due to pressure.[5]

Mineral imbalances could conceivably exist in the rations of many herds from which these cases originated. Increased solar radiation at these altitudes could also play a part in the production of more than optimal amounts of vitamin D; white muscle disease, believed due to vitamin E

deficiency, is also occasionally observed in the area. If increased portal pressure alone could initiate these changes in the vessels, their appearance would undoubtedly be recognized more frequently.

Because of the consistent hepatic changes, interest might be centered in their development aside from any direct effect on the brisket disease syndrome. The following microscopic changes have been described in chronic passive congestion of the liver: capillary dilatation with atrophy of the central cells, central degenerative changes, central fat accumulation, central necrosis associated with hemorrhage or fatty infiltration, and central fibrosis. These changes are all associated with different forms of circulatory disturbance. Necrosis of the liver cells in chronic passive congestion is due to stasis, with asphyxia of cells farthest from arterial blood.[10] Chronic passive congestion of the liver has been produced by partial ligation of the thoracic portion of the inferior vena cava, followed by central hemorrhage, necrosis, and cirrhosis of the liver, with collection of pleural and ascitic fluids.[23] Blood flow obstruction has been associated with cirrhosis with resultant changes in splenic vessels, passive congestion of the digestive tract, and escape of fluid into the peritoneal cavity.[13] Central hepatic fibrosis has been found directly related to congestive heart failure. Other types of fibrosis, except biliary, were more frequent with congestive heart failure but were not confined to it. Chronic passive congestion with anoxia may thus increase the susceptibility of hepatic tissue to injury in both central and portal areas.[9]

Chronic passive congestion could thus be considered the chief cause of the central changes in the liver lobule, proceeding from central congestion to final stages in which fibrosis develops. With extensive change, another impediment to the circulation might result, aggravating the results of a vicious cycle. Proliferation of the bile ducts would seem to be of other than circulatory failure origin and, while periportal changes could be ascribed to chronic passive congestion, they are frequently associated with toxic manifestations.

In blind staggers and alkali disease from selenium intoxication, many lesions in common with brisket disease syndrome were observed, including sinusoidal congestion, necrosis, bile duct regeneration, and periportal fibrosis of the liver. Cardiac decompensation appeared in the chronic form of alkali disease.[6] Rats placed on experimental diets containing selenium developed hepatic cirrhosis with ascites and anemia. Fibrosis was often periportal.[11] Rabbits fed seleniferous oats also developed pleural and peritoneal effusions and liver scarring.[22] Steers kept on seleniferous range for two years were found to have 2.2 p.p.m. of selenium in the kidneys and 1.2 p.p.m. in the liver; steers on such range for three years had values of 4.1 and 5.7, respectively.[17]

Cardiac decompensation and associated changes, bile duct regeneration, and periportal hepatic fibrosis were also seen in this series of brisket syndrome cases. In 9 of these animals, suggestive levels of selenium were found. Sections from 2 were unavailable for study but, of the remaining 7, a thickened liver capsule was found in 6; central congestion surrounded by a layer of fatty infiltration and then by normal liver parenchyma in 4; periportal fibrosis in 3; a combination of central fatty metamorphosis, hemorrhage, necrosis, or fibrosis in 2; and central fatty infiltration in 1. While definite, toxic levels of selenium are unavailable for diagnostic purposes as they are for metallic poisons, reproduction of similar lesions in laboratory animals with seleniferous diets strongly indicates selenium may be a possible factor in at least some cases of brisket disease syndrome.

Increased resistance to the circulation through the lungs due to atmospheric hypoxia, chronic alveolar emphysema, arteriosclerosis of the pulmonary vein, or pneumonia has been reported responsible for cardiac failure developing the signs associated with brisket disease syndrome.[18]

While the importance of pneumonic changes as an etiological factor can not be discounted, the effect of pathological changes in other organs should also be considered since hepatic changes, even where secondary, might further interfere with the circulation. Renal changes, while often minor, were sometimes suggestive of those changes associated with human hypertension.

As pointed out in the discussion of liver changes, many of the hepatic changes in man can be attributed to cardiac disease. While valvular lesions were never extensive, their recognition in 36 per cent of

these cases suggests a direct bearing on the production of brisket disease syndrome. The myocardial necrotic foci and thrombi seen in 4 cases would also appear to be important causative factors. The cardiac decompensation associated with selenium poisoning might be a primary effect of the poisoning, although it has not been reported in studies with laboratory animals.

While cause may be separated from effect with difficulty, the possibility that a variety of factors, possibly working together in many instances, may be responsible for brisket disease syndrome should not be overlooked.

SUMMARY

The gross and microscopic changes of approximately 50 cows with brisket disease syndrome are described. It is suggested that a variety of conditions, such as anemia from anaplasmosis, arteriosclerosis, and damage to the major organs and heart may operate in conjunction with the stress of altitude in producing this syndrome.

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Impacted Colon in Young Pigs.—Impaction (coprostasis) was found in 21 (20%) pigs less than 2 weeks old, in a series of necropsies in Norway. The affected pigs averaged 7 days old and none were more than 2 weeks old. The obstruction was invariably in the ventral loop of the spiral colon (the last part of the centripetal, and the first part of the centrifugal colon). The obstructing mass was usually barley beards with their barbs caught in the mucosa. Therefore, barley chaffs should not be used as bedding for young pigs.—*Otto Roneus in Nord. Vet.-med. (May, 1957): 362.*

Cattle Grub Eradication

The development of systemic insecticides or "grubicides" (*see* page 421) presents a challenging opportunity for the livestock, meat, and leather industries to embark on a program designed to eradicate one of the most damaging parasites affecting cattle and beef production—the cattle grub or warble.

Since the damage inflicted by the grubs appears to affect all of these industries, it is not surprising to find them almost unanimously supporting programs that may reduce their losses.

The livestock producer observes these parasites both as "heel" flies which cause much anxiety and characteristic running by the frantic cattle, and as grubs which produce swellings in the backs of animals during the late winter and spring. To the meat packer, they mean a loss from trimming and subsequent devaluation of carcasses, and to the leather industry "grubby hides" and "grub discounts" mean adding burdens to a product (leather) which is already constantly subjected to the economic pressures of synthetic substitutes.

Efforts to control or eliminate this parasite have been sporadic, partly due to the fluctuations in the values of damaged products but, to a larger extent, due to the failure of recommended control procedures to meet the test of effectiveness and practicability.

The past programs of cattle grub control, using rotenone or other local applications, could be aptly described as "too little and too late." The damage had already been done and the hope lay only in preventing development of a crop of warble flies the next season. Even with the best cooperative effort toward area control, with agents designed to kill the larvae after they appeared in the backs of cattle, results were disappointing.

The new methods of grub control (the systemic chemicals given orally) introduce a new concept of parasite control.

Anticipated Problems

The first systemic grubicide to receive approval for wide use is an organic phosphorus compound, DOW ET-57 (Trolene).

This product is not entirely without danger in so far as toxicity to livestock is concerned (*see* JOURNAL, March 1, 1957: 215). In addition, its residues are found in milk and meat of animals for periods of at least 30 days following treatment. Therefore, since no permissible tolerances have been established for this chemical in food products, its use in livestock is predicated on the complete absence of residues in the food products. This precludes its use on dairy cows and on animals which are to be slaughtered for food purposes within 60 days following treatment.

The problem of toxicosis in man from pesticides has received continuing attention in medical circles (*J.Am.M.A.*, April 13, 1957: 1338).

Pesticides are being used, in the United States, in about 80,000 preparations with over 200 basic chemicals. Human deaths ascribed to accidental poisonings by pesticides have ranged, in recent years, from a high of 151 in 1946 to a low of 104 in 1952. In about 50 per cent of these cases, death has been ascribed to arsenic compounds but, in from 10 to 20 per cent, the poison has remained unidentified.

Whether precautionary labeling of pesticides is an adequate safeguard for both livestock and the public is debatable, particularly when the economic pressures for, and popularity of, mass treatments of livestock are so strong.

Even though the veterinary profession is well represented on the newly created National Cattle Grub Committee of Livestock Conservation, Inc. (*see* page 434), the question of how the veterinary practitioner fits into an organized effort to eradicate cattle grubs needs to be explored. Too much is at stake—public safety, livestock health, and economic gains—to assume that veterinarians will not become involved in a major way in such an undertaking.

The service that the individual veterinarian can render in this program will depend to a large extent on (1) his knowledge of the drugs being employed; (2) his ability to differentiate between their side effects and manifestations of other diseases; and (3) on his initiative in helping to solve the residue problem and in advancing a worthy project.

Current Literature

ABSTRACTS

Heat-Inactivated Vesicular Stomatitis Virus

Suspensions of bovine tongue epithelium containing New Jersey or Indiana types of vesicular stomatitis virus (VSV) were heated at 56 and 60 C. for 30 minutes. Dilutions of these suspensions were inoculated into suckling mice, embryonating chicken eggs, guinea pigs, cattle and tissue cultures. In all cases described, heating rendered the virus suspensions noninfective but did not significantly affect their serological activity or impair their usefulness otherwise.—[*G. T. Dimopoullos, O. N. Fellowes, J. J. Callis, G. C. Poppensick, J. Tessler, and W. R. Hess: Heat-Inactivated Vesicular Stomatitis Virus as Antigen in the Complement-Fixation Test. Am. J. Vet. Res., 18, (July, 1957): 688-690.*]

Ringworm in Wild Animals in Southwestern Georgia

In a survey of 1,142 wild animals of 21 species for ringworm, dermatophytes were isolated from hair specimens of 88 (7.7%) of the animals. The organisms commonly isolated were *Microsporum gypseum* from *Peromyscus polionotus* and *Rattus norvegicus; M. gypseum* ("red variety") from *Reithrodontomys humulis, P. polionotus, Peromyscus gossypinus, Peromyscus nuttalli, Mus musculus, Sigmodon hispidus,* and *Neotoma floridana;* and *Trichophyton mentagrophytes* from *Didelphis virginiana, P. polionotus, P. gossypinus, M. musculus, S. hispidus, R. norvegicus,* and *Rattus rattus.* Infection was more common in young rats (12.5%) than in old rats (4.2%), and there was little difference in the infection rate between the sexes. The seasonal peak (13.5%) occurred during the spring months. The peak month for all of the species was in April (20.8%) with another lower peak (13.0%) in July. The distribution of infection was similar for the four counties where most of the animals were trapped. Sixty-two soil specimens, from areas where animals were trapped, were cultured and *M. gypseum* was isolated from 16 (26.0%).—[*R. W. Menges, G. J. Love, W. W. Smith, and L. K. Georg: Ringworm in Wild Animals in Southwestern Georgia. Am. J. Vet. Res., 18, (July, 1957): 672-677.*]

Genital Vibriosis in Sheep

A number of abortions (18) due to vibriosis occurred in a herd of 142 breeding ewes. In addition to those isolated from aborted fetuses, vibrios were isolated from (*a*) vaginal mucus from pregnant ewes which produced healthy lambs; (*b*) vaginal mucus from virgin ewes; (*c*) fetal fluid from normally lambing ewes; (*d*) gastric contents from normal lambs at birth; and (*e*) semen from the rams. All Vibrio strains isolated reacted similarly to: catalase activity, H_2S-production, nitrate reduction, and stab culture tests. The abortifacient effect on pregnant guinea pigs was similar for all strains.

Cross-agglutination tests indicated serological relationships between the strains. A noncoital route of infection was more important than a coital one. Obviously, the infection was spread among the sheep mainly after appearance of abortion. Therefore, immediate measures for isolation and disinfection are important. The cattle herd in contact with the Vibrio-infected sheep showed no signs of the infection.—[*Gunnar W. Eide and Oddvar Helle: Studies on Genital Vibriosis in Sheep. Am. J. Vet. Res., 18, (Oct., 1957): 868-872.*]

Identification of Strychnine and Other Alkaloids

A relatively simple and rapid standardized procedure for the isolation and identification of alkaloids, particularly strychnine, from animal tissues has been developed. It involves the extraction of the alkaloid from tissue or stomach contents by acid or an acid-alcohol mixture, preliminary testing on paper and spot-plates, circular paper chromatography, and ultraviolet spectrophotometry.—[*Dennis H. Cox: Isolation and Identification of Strychnine and Other Alkaloids in Veterinary Toxicology. Am. J. Vet. Res., 18, (Oct., 1957): 929-932.*]

Maltase Activity in Canine Blood Serum

A method for assaying the maltase activity of dog serum based on increased reducing power of a phosphate buffered maltose solution containing 0.01 ml. of dog serum per 8 ml. of solution is described. Incubation was at 39 C. Optimal pH was found to lie between pH 6.4 and 6.6, although strong activity was present between pH 6.1 and 7.3. The effects of varying concentrations of maltose were studied at pH 6.6. Maximal hydrolysis occurred at a maltose concentration of approximately 3.5×10^{-3} M. Higher concentrations showed decreasing activity until complete inhibition occurred at about 8.0×10^{-2} M. The maltase activity of the serums of 15 healthy dogs was determined using a maltose concentration of 7.14×10^{-4} M at pH 6.6. The average hydrolysis by 0.01 ml. of these serums was 1.75×10^{-6} M. of maltose in 60 minutes. The range was from 1.18×10^{-6} to 2.48×10^{-6} moles.—[*R. G. Skillen: Determination of Maltase Activity in the Blood Serum of the Dog. Am. J. Vet. Res., 18, (July, 1957): 638-641.*]

The Intradermal Sarcocystin Test

The paper describes the experimental use of a skin test in the diagnosis of sarcosporidiosis. The antigen was extracted by freezing and thawing of macroscopic sarcocysts from sheep oesophagi. The test dose of the antigen was estimated to be 0.1 ml. of 1:200 dilution. A test was regarded as positive when an area of erythema and edema measuring 1.0 to 2.0 cm. in diameter was present at the end of 48 hours. The reaction was of the delayed type.

A comparison of results from the skin test with a dye test and CF test indicated that there is a correlation between the positive reactors to the skin test and the dye test but not with the CF test.

The author concluded that the postive reactions to the skin test often occur in the absence of established infection. While this test has little specific diagnostic value, it can be used as a screening test.—[*F. I. Awad: The Intradermal Sarcocystin Test. Am. J. Vet. Res., 18, (July, 1957): 703-704.*]

FOREIGN ABSTRACTS

Feeding Pups

This paper includes a brief review of previously published work on various formulas for feeding pups. The experiments with different formulas are described and the results are presented in tables and graphs showing the growth curves. The following formula is recommended: 800 ml. of whole milk mixed with 200 ml. of cream (12% butterfat), one egg yolk, 6 Gm. of bone meal, and 4 Gm. of citric acid dissolved in a small amount of water. The formula should have a total of about 2,000 units of vitamin A and 500 units of vitamin D which may be obtained by using one of the commercially available vitamin preparations.

Pups are fed six times a day for the first three weeks by means of an ordinary nursing bottle. For small pups, one may use a small rubber bulb from a medicine dropper. After three weeks, they can be fed from a dish and the number of daily feedings can be reduced to five. The formula need be used for only four weeks, after which they can be given solid food.

The amount of formula to give depends on the age. Pups should not be force fed. They should take enough so that the stomach can be barely palpated back of the costal arch. Pups of breeds of average size, at 3 days of age, should be fed daily an amount equal to about 15 to 20 per cent of their body weight. Pups 3 weeks old may take a daily amount equal to 35 per cent of their weight.—[*Björck, G., Olsson, B., and Dyrendahl, S.: Artificial Feeding of Puppies. Nord. Vet.-med., 9, (1957): 285-304.*]—ALFRED G. KARLSON

Renal Cortical Hypoplasia in the Dog

Forty cases of renal cortical hypoplasia have been seen in Cocker Spaniels during the period 1946-1956. Males and females were equally involved. The ages ranged from 2 months to 4 years, with most being over 12 months of age. The disease appears to have an hereditary basis. Presenting symptoms consisted of inappetence, loss of weight, vomition, polyuria, and polydipsia. The urine usually had albumin, an acid reaction, and low specific gravity. Azotemia was always present. In the only dog examined for calcium and phosphorus, the blood levels were high. The animals were small for their age, reminding one of renal dwarfism in man. The kidneys were small and had streaks of calcium in the medulla and occasionally foci of calcium in the cortex.

In all cases, there was hypertrophy of the left side of the heart and dilatation of the right side. There was usually pulmonary edema and congestion of the liver. The parathyroid glands were always enlarged. Calcinosis was noted in the endocardium, parietal pleura, vocal cords, and gastric mucosa. In a few cases, there were obvious bone changes such as softening of the frontal bone, both upper and lower jaws, and ribs.

Microscopically, the kidneys showed severe sclerosis and calcinosis. The latter was found in various organs. The parathyroid gland consisted almost entirely of light goblet cells. The bones showed fibrous osteodystrophy, which was most pronounced in the mandible. The condition results from a vicious cycle between the hypoplastic kidney and the hyperfunctioning parathyroid as follows: The low activity of the kidney leads to hyperphosphatemia which, in turn, results in hypocalcemia and hyperparathyroidism. The increased activity of the parathyroid gland results in calcinosis and osteodystrophy.—[*Lennart Krook: The Pathology of Renal Cortical Hypoplasia in the Dog. Nord. Vet.-med., 9, (1957): 161-176.*]—ALFRED G. KARLSON.

The Whiteside Test for Mastitis

A review of pertinent literature is followed by a description of the author's experience with the Whiteside test on pooled milk during a four-year period. It was concluded that the Whiteside test provides information that correlates well with other bacteriological and biochemical tests for mastitis. The Whiteside test proved to be better than the catalase test in revealing herds with mastitis. Bacteriological examination using Staphylococcus toxin-blood agar proved to be of least value in detecting infected herds.—[*F. Anderson: Investigations on the Use of the Whiteside Test for Mastitis. Nord. Vet.-med., 9, (1957): 191-209.*]—ALFRED G. KARLSON.

BOOKS AND REPORTS

Clinical Toxicology of Commercial Products

With the numerous chemical products available on the market today (insecticides, pesticides, cleansers, and others), there is an increased danger of accidental poisoning, especially of children and animals. This volume is designed to make urgently needed information immediately available in the home and on the farm as well as to other agencies (pharmacies, industrial medical departments, public health nursing centers, etc.) which might be called upon for emergency help.

The book is divided into seven sections covering first aid and general emergency treatment; indexes of chemical ingredients, therapeutic agents, and trade-name products; supportive therapy; general formulas of chemical agents; and manufacturers' names and addresses. These various sections are printed on different colored paper for quick reference.—[*Clinical Toxicology of Commercial Products. By Marion N. Gleason, Robert E. Gosselin, and Harold C. Hodge. 1,160 pages. Williams and Wilkins Co., Mt. Royal and Guilford Ave., Baltimore 2, Md. 1957. Price $16.00.*]

THE NEWS

National Cattle Grub Committee Completed

Dr. J. W. Cunkelman, chairman, National Cattle Grub Committee, Livestock Conservation, Inc., has announced the completion of four committees to review and study grub control measures. This national committee was recently established at the cattle grub workshop held in Kansas City under the sponsorship of Livestock Conservation, Inc. The committee selections announced by Dr. Cunkelman are:

Research and Evaluation.—E. F. Knipling, director, Agricultural Research Service, U.S.D.A., chairman; Harry Boyts, manager, Sioux Livestock Conservation, Inc.; Dr. Fred O'Flaherty, Tanners' Council of America; John A. Killick, National Independent Meat Packers Association; Dr. L. E. Swanson, American Association of Veterinary Parasitologists; and Jack Dreessen, National Agricultural Chemicals Association.

Economics of Grub Control.—Roy Lilley, assistant secretary, American National Cattleman's Association, chairman; Jim Rosse, manager, Omaha-Denver Livestock Conservation, Inc.; Cecil Powell, Tanners' Council of America; L. E. Taylor, American Meat Institute; Earl McQuown, Corn Belt Livestock Feeders Association; and Karl B. Musser, secretary, Purebred Dairy Cattle Association.

Education and Information.—C. E. Bell, chief, Animal Industry Branch, Federal Extension Service, U.S.D.A., chairman; Doug Mossberg, manager, Northwest Region, Livestock Conservation, Inc.; Dr. C. M. Patterson, Extension Veterinarian, Texas A. & M.; Blaine Liljenquist, Western States Meat Packers Association; and Frank Richards, secretary, Aberdeen-Angus Association.

Program and Procedure.—Herman Aaberg, assistant commodity director, American Farm Bureau Federation, chairman; Ray Cuff, manager, Southwest Region, Livestock Conservation, Inc.; Dr. John Herrick, Extension Veterinarian, Iowa

General Kester Retires from the Air Force

The Air Force recently announced the retirement of Brig. Gen. Wayne O. Kester, U.S.A.F. (V.C.), First Assistant for Veterinary Services to the Air Force Surgeon General, effective Aug. 31, 1957. During ceremonies at Air Force Headquarters, General Kester was presented the Legion of Merit (First Oak Leaf Cluster) by Major General Dan C. Ogle, U.S.A.F. Surgeon General, awarded by the President for exceptionally meritorious conduct in the performance of outstanding services, and a citation by the chief of staff for contributions to the development of the Air Force. The ceremonies were held in General Ogle's headquarters and were attended by Mrs. Kester and the senior staff of the surgeon general.

General and Mrs. Kester plan to remain in Washington where he is president of the Microbiological Foundation and executive vice-president of Microbiological Associates, Inc., 4846 Bethesda Ave., Washington 14, D.C.

Pictured left to right—General Ogle, General Kester, and Mrs. Kester.

State College; W. D. Knox, editor, Hoard's Dairyman; John Macfarlane, field director, New England Livestock Conservation, Inc.; Dr. M. R. Clarkson, deputy administrator, Agriculture Research Service, U.S.D.A.; and Dr. E. E. Saulmon, assistant to director, ARS, Animal Disease Eradication Division, U.S.D.A.

A.M.A. Council on National Defense Sponsors Eighth Annual Conference

The Council on National Defense of the American Medical Association is sponsoring the eighth annual conference of the County Medical Societies Civil Defense Organization. The conference will be held at the Morrison Hotel, Chicago, on Nov. 9-10, 1957.

The conference program is planned to acquaint individuals, at local medical and health group levels, with the results and recommendations of several operational test exercises which have been conducted under simulated disaster conditions. One of the features of the conference is a comprehensive report on "Hurricane Audrey 1957."

Individuals are provided the opportunity during the conference to discuss and exchange information concerning emergency medical activities and to participate in workshop sessions dealing with civil defense affairs. A $12 registration fee is charged to defray the costs of the two luncheons and other administrative expenses.

The Morrison Hotel, 79 W. Madison St., Chicago, has set aside a block of rooms for the convenience of the conferees. Individuals are urged to contact the hotel directly for reservations, making mention of the conference.

Those desiring additional information about the conference are requested to contact Mr. Frank W. Barton, secretary, Council on National Defense, American Medical Association, 535 N. Dearborn St., Chicago 10.

AMONG THE STATES AND PROVINCES

Arkansas

Dr. R. P. Scott with Arkansas Livestock Sanitary Board.—Dr. Joseph P. Scott (OSU '14) retired from his position with Jensen-Salsbery Laboratories in 1956, and is now with the Arkansas Livestock Sanitary Board at the Russellville Poultry Disease Laboratory.

Dr. Scott had been with Jen-Sal Laboratories for 12 years prior to his retirement.

Alabama

Central Alabama Association.—The officers of the Central Alabama V.M.A. for the coming year are as follows: Dr. G. J. Phelps, Jr., Montgomery, president; Dr. B. N. Lauderdale, Montgomery, vice-president; and Captain J. T. Williams, U.S.A.F. Hospital, Maxwell Air Force Base, Ala., secretary-treasurer.

s/JOE T. WILLIAMS, *Secretary.*

Illinois

Chicago Association.—The regular monthly meeting of the Chicago V.M.A. was held on Tuesday, Sept. 10, 1957, at 8:30 p.m. at the LaSalle Hotel.

Dr. Thomas Powers, College of Veterinary Medicine, Ohio State University, spoke on "New Chemotherapeutic Agents in Small Animal Practice."

s/MARK DAVENPORT, *Secretary.*

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Northern Illinois Association.—The thirty-seventh annual meeting of the Northern Illinois V.M.A. was held on Wednesday, Sept. 18, 1957, at the Hotel Faust in Rockford. The registration included 137 members and guests.

The speakers on the program and their subjects included: Dr. A. R. Skewes, practitioner, Union Grove, Wis.—Horse and Pony Practice; Dr. T. H. Brasmer, practitioner, Danville—Emergency Surgery of the Lung and Abdomen; and Dr. A. K. Merriman, superintendent, Division of Livestock Industry, Springfield, and Dr. Edward J. Brown, veterinarian in charge, Illinois Animal Disease Eradication Division, Springfield—State and Federal Regulations.

The afternoon program consisted of the following: Dr. Charles J. York, Virus Research Laboratories, Pitman-Moore Co., Indianapolis, Ind.—Infectious Bovine Rhinotracheitis; Mr. Wilbur Plager, field secretary, Iowa Swine Growers Association, Des Moines, and Dr. D. W. Rawson, practitioner, Milledgeville—Problems of the Swine Industry Today; and Mr. Ray Morris, attorney, Illinois State V.M.A. legal councilor, Chicago—Report of State Legal Councilor. The local arrangements for this meeting were made by Drs. B. L. Lake and D. R. Stephenson, both of Rockford.

The officers elected for the coming year are: Drs. J. M. Nelson, DeKalb, president; J. G. Hardenbergh, Rockton, president-elect; D. W. Rawson, Milledgeville, secretary-treasurer.

s/J. G. HARDENBERGH, *President-Elect.*

Indiana

Central Indiana Association.—The Central Indiana V.M.A. met at the Italian Village, Sept. 9, 1957. Dr. Virgil Robinson, Indianapolis, presented a paper on the condition of pancreatitis as it is seen in large and small animals.

The officers elected for the ensuing year are: Drs. John Templeton, McCordsville, president; Charles Stengel, Indianapolis, vice-president; and Peter Johnson, Indianapolis, secretary.

Iowa

Cedar Valley Association.—The Cedar Valley V.M.A. held its first meeting of the year in Waterloo, Sept. 9, 1957. Forty-two members attended.

The topic, "Current Field Problems," was discussed by Drs. C. K. Pfaff, Fort Dodge Laboratories; C. D. Lee, Iowa State College; W. M. Lynch, Allied Laboratories; and G. H. Gitz, U. S. Department of Agriculture.

s/A. J. COTTEN, *Secretary.*

Kansas

Kansas City Association.—The September meeting of the Kansas City V.M.A. was held in Exchange Hall, Livestock Exchange Bldg., Sept. 17, 1957.

Large and small animal sections were combined on the program. An Upjohn film showing the latest techniques in surgery entitled, "Abdominal Surgery," was also shown.

s/R. E. GUILFOIL, *Secretary.*

Massachusetts

Eastern States Exposition.—The Massachusetts Veterinary Association sponsored a closed circuit telecast in conjunction with the association's exhibit at the Eastern States Exposition in Springfield, Mass., Sept. 14-22, 1957. This program was made possible through Mr. L. T. Maloney, public relations consultant, in cooperation with Chas. Pfizer & Co., Inc., Department of Veterinary Medicine.

There were almost 100 hours of telecasting in 12 strategic spots at the exposition, comprising three- to five-minute programs from 11:00 a.m. to 8:00 p.m., daily, during the nine days of the display.

This was a unique opportunity to project the veterinary profession's activities and goals, and those of New England veterinarians in particular, before the thousands of people who saw the exhibit and who were largely unaware of the many facets of the veterinarian's role in society.

Michigan

Dr. Gail E. Hawley Scholarship Presented to Michigan State University.—A $5,000 scholarship fund for veterinary medical students at Michigan State University has been accepted by the State Board of Agriculture.

The fund is a gift from Mrs. Georgia E. Corbin, Grand Rapids, in memory of her son, Dr. Gail E. Hawley, a 1950 graduate of the College of Veterinary Medicine, Michigan State University. Dr. Hawley died March 16, 1957.

The scholarship to be awarded from the fund will be known as the "Dr. Gail E. Hawley Scholarship," and will consist of $500 each year to a student in the fourth year of study in the College of Veterinary Medicine, according to Dr. Thomas H. Hamilton, vice-president for academic affairs.

To qualify for the scholarship, students must be above average in scholarship, have financial need, and give promise of making a contribution to the profession of veterinary medicine.

New York

Veterinary Medical Association.—The regular meeting of the Veterinary Medical Association of New York City, Inc., was held on the evening of Oct. 2, 1957, at the New York Academy of Sciences, New York City.

Dr. E. E. Ruebush, Ambassador Animal Hospital, Silver Springs, Md., spoke on "The Practical Application of the Electrocardiograph in Small Animal Medicine."

s/C. E. DECAMP, *Secretary.*

Ontario

Ernst Leitz Fellowship Awarded to Dr. James Archibald.—Dr. T. Lloyd Jones, principal, Ontario Veterinary College, Guelph, has announced the award of the Ernst Leitz Fellowship to Dr. James Archibald, Department of Medicine and Surgery.

The fellowship is awarded by the Ernst Leitz Company of Wetzlar, Germany, to graduates of the Ontario Veterinary College for advanced study at the University of Giessen in West Germany.

Dr. Archibald will be accompanied by his wife, Dr. Hilda Corless Archibald, and their two sons. On completion of his studies at the University of Giessen, he will serve as an exchange professor at the Veterinary School, University of Glasgow. Dr. Archibald and his family will be in Europe for one year.

Oregon

Oregon Auxiliary Meeting.—The fall meeting of the Women's Auxiliary to the Oregon V.M.A. was held at Tillamook on Sept. 13, 1957. The business meeting was held during a luncheon in the Panarama Room at nearby Rockaway Beach. Of much interest was the announcement that our immediate past-president, Mrs. Austin Eivers of Salem, had been elected as recording secretary of the Women's Auxiliary to the AVMA at the convention held last August in Cleveland.

Mrs. Earl Derflinger, delegate to the Auxiliary house of representatives, gave an enlightening report on the meeting in Cleveland.

In the evening, the wives joined their husbands on the beach for a barbecued crab feed.

s/MRS. DALLEN H. JONES, *Secretary.*

Quebec

Dr. André Lagacé Receives Ph.D. Degree.—Dr. André J. Lagacé (MON '54), professor, College of Veterinary Surgeons of the Province of Quebec, has completed his postgraduate studies at the College of Veterinary Medicine, Ohio State University, on "The Toxic and Therapeutic Effects of Stilbamidine and Hydroxystilbamidine in the Blastomycosis of the Dog." Dr. Lagacé, on a research fellowship from the Agricultural Research Council, Department of Agriculture of the Province of

Quebec, worked under the direction of Dr. C. R. Cole, professor of pathological anatomy at Ohio State University.

Vermont

New England Association.—The New England V.M.A. held its twenty-third annual meeting at Equinox House in Manchester, Vermont, Oct. 6-9, 1957. The veterinarians from Vermont and their wives were co-hosts to the members and ladies of the New England V.M.A.

Among the speakers and subjects on the program were: Drs. W. J. Gibbons, Auburn, Ala.—Forage Poisoning; R. W. Kirk, Ithaca, N. Y.—Infectious Diseases of Cats; Robert A. Sauter, Omaha, Neb.—A New Killed Rabies Vaccine for Animals; and W. W. Armistead, East Lansing, Mich.—Small Animal Surgery (illustrated).

On the third day of the meeting, a movie depicting the atom bomb tests was shown. A selection of the veterinarians who spoke October 8, and their topics are as follows: Drs. M. G. Fincher, Ithaca, N. Y.—Metabolic Diseases of Cattle; Bernard F. Trum, Washington, D. C.—The Veterinarian's Role in Atomic Radiation; Dale G. Friend, Boston, Mass.—Tranquilizing Drugs; and Jacques Jenny, Philadelphia, Pa.—Modern Trends in the Treatment of Bone and Joint Injuries.

Dr. W. W. Armistead, president of the AVMA, dean, College of Veterinary Medicine, Michigan State University, extended "Greetings From AVMA" at the annual banquet and Mrs. A. E. Coombs presented the "Auxiliary Greetings."

Washington

FOREIGN NEWS

Dr. Kaschula Returns to South Africa.—Dr. Victor R. Kaschula, who has been on the staff of the veterinary department, Rutgers University, New Brunswick, N. J., for the past few years, has accepted a position with the United States Foreign Aid Program, where he will study the ecology of diseases of animals in Northern Nigeria for the next two years. Many will remember his fine discussions of exotic diseases at the Regional Conference sponsored by the U. S. Department of Agriculture during March and April of 1956.

U. S. GOVERNMENT

Veterinary Personnel Changes.—The following changes in the force of veterinarians in the U.S.D.A. are reported as of August 28, and Sept. 4, 9, and 11, 1957.

TRANSFERS

John C. de Holl, from Indianapolis, Ind., to Sioux City, Iowa.
Arnold V. Giesemann, from St. Louis, Mo., to Chattanooga, Tenn.
Charles J. Koerth, Jr., from Yakima, Wash., to Scottsbluff, Neb.
Harold A. Krig, from Madison, Wis., to Bismarck N. Dak.
George Migaki, from Portland Ore., to Beltsville, Md.
Lyle V. Russell, from Omaha, Neb., to Billings, Mont.
Jerry R. Steffen, from St. Joseph, Mo., to Des Moines, Iowa.
Gilbert H. Wise, from Trenton, N. J., to Columbus, Ohio.

RETIREMENTS

Byron W. Beckett, Cincinnati, Ohio.
Carl Vernon Metcalf, Kansas City, Kan.
Harry L. Shorten, Denver, Colo.

A U.S.D.A. Unit Award for Superior Service was presented to the Animal Disease Eradication Branch of ARS, Olympia, Wash., for cooperating with the State of Washington in organizing and servicing the Brucellosis Eradication Program so effectively that statewide certified brucellosis-free area status was achieved four years before national certification.

From left to right—Dr. E. E. Saulmon, assistant to director, Animal Disease Eradication Division, ARS, U.S.D.A.; Dr. George Duby, president of the Washington State V.M.A.; Mr. Charles Figy, assistant to the Secretary of Agriculture; Dr. F. M. Shigley, inspector in charge, ARS, U.S.D.A.; Dr. A. E. Crouse, assistant director, Washington State Department of Agriculture; and Dr. Irwin Erickson, past-president, Washington State V.M.A.

DEATHS

Coleman P. Callaway, San Antonio, Texas.
Walford J. Johnson, Sioux City, Iowa.

STATE BOARD EXAMINATIONS

COLORADO—Jan. 15-16, 1958, Arvada; and first week in June, depending on graduation date. Fort Collins. W. D. Stauffer, secretary, 5500 Wadsworth Blvd., Arvada, Colo.

ILLINOIS—Dec. 4-6, 1957. Chicago. F. B. Selcke, registration superintendent, Springfield, Ill.

MINNESOTA—Jan. 6-7, 1958, St. Paul. A. C. Spannaus, secretary, Route 1, Waconia, Minn.

NEW JERSEY—Dec. 27-28, 1957, Trenton. Joseph A. S. Millar, secretary, Box 172, Deal, N.J.

NORTH DAKOTA—April 9-10, 1958, Fargo. M. C. Hawn, secretary-treasurer, 1407 13 St. N., Fargo, N. Dak.

NEW MEXICO—Jan. 13, 1958, Santa Fe. Edwin J. Smith, secretary, P.O. Box 4385, Santa Fe, N.M.

TEXAS—Jan. 15-17, 1958, Austin. T. D. Weaver, executive secretary, 207 Capital National Bank Building Austin 16, Texas.

DEATHS

Star Indicates Member AVMA

Ernest R. Bush (CIN '15), 65, died in Dallas, Texas, on March 27, 1957, after a long illness.

★**Harry E. Breckerbaumer** (ISC '08), 75, died unexpectedly in Hollywood, Calif., Aug. 13, 1957.

Dr. Breckerbaumer had been a meat inspector at the Sioux City stockyards for 42 years before his retirement in 1950. At the time of his death, he was in California on vacation with his daughter, Mrs. Louise Walker, Phoenix, Ariz. He is a member of various lodges in Sioux City, Iowa, as well as the National Association of Federal Veterinarians, the High Twelve, National Association of Retired Civil Service Employees, and the National Association of B.A.I. Veterinarians.

★**Benjamin Franklyn Gooch** (KVC '07), 73, Kansas City, Mo., died on March 8, 1957. Dr. Gooch was employed by the federal government for 43 years before he retired in 1949.

He was a member of the Veterinary Reserve Corps and the National Association of B.A.I. Veterinarians.

Ralph F. Graham (ISC '04), 74, Kaw City, Iowa, died July 1, 1957, at the Ponca City Hospital. He had been active in the management of his 10,000-acre ranch up to a few days before his death.

Dr. Graham is survived by his widow, a son, a daughter, and two brothers.

★**William David James** (IND '16), 62, Greencastle, Ind., died on June 1, 1957.

Dr. James had practiced veterinary medicine in Greencastle for more than 20 years and he formerly had served as the town's mayor. He was also a member of the Indiana V.M.A.

★**William J. Lee, Sr.** (UP '09), 72, formerly of Philadelphia, Pa., died Aug. 15, 1957.

Dr. Lee was a former professor of veterinary surgery at the University of Pennsylvania until his retirement ten years ago. He had been associated with the veterinary school faculty since his graduation, first as a demonstrator and instructor, later as assistant professor and associate of the late Dr. John R. Adams whom he succeeded as professor after the latter's death., and up until the time he retired in 1947.

Dr. Lee specialized in equine surgery and was well known in Thoroughbred racing and horse show circles.

Survivors include his widow, four daughters, and three sons, two of whom are veterinarians: Dr. John M., and Dr. William J., Jr. The third son, Dr. Jeremiah F., is a physician.

★**George W. Musselman** (MCK '11), 79, Denver, Ind., died at his home Aug. 17, 1957, after a three-year illness.

Dr. Musselman had practiced veterinary medicine in Denver for 44 years before he retired in 1955. He was an honorary member of the Indiana V.M.A.

He is survived by his widow, Jessie, a son, and a daughter.

★**John Reichel** (UP '06), 71, onetime director of laboratories of the H. K. Mulford Co., Glenolden, Pa., later the owner of Reichel Laboratories, Kimberton, Pa., died Aug. 16, 1957.

Earl W. Sailor (STJ '21), 66, Grand Junction, Iowa, died Aug. 12, 1957, after suffering a heart attack.

A veterinarian in Grand Junction for many years, Dr. Sailor had been working recently as a U. S. poultry inspector at Atlantic, Iowa.

Dr. Sailor is survived by his wife, two brothers, and a sister.

★**Edrick K. Tingley** (UP '13), 70, formerly of Marietta, Pa., and onetime president of Gillitand Laboratories, died July 15, 1957, in Elizabethtown, Pa., where he and his wife had been living since Dr. Tingley retired a number of years ago.

• • •

Other Deaths Reported.—The following deaths have been reported. The usual information for an obituary was not supplied.

Milton I. Brown (ISC '23), Perry, Iowa, died on July 1, 1957.

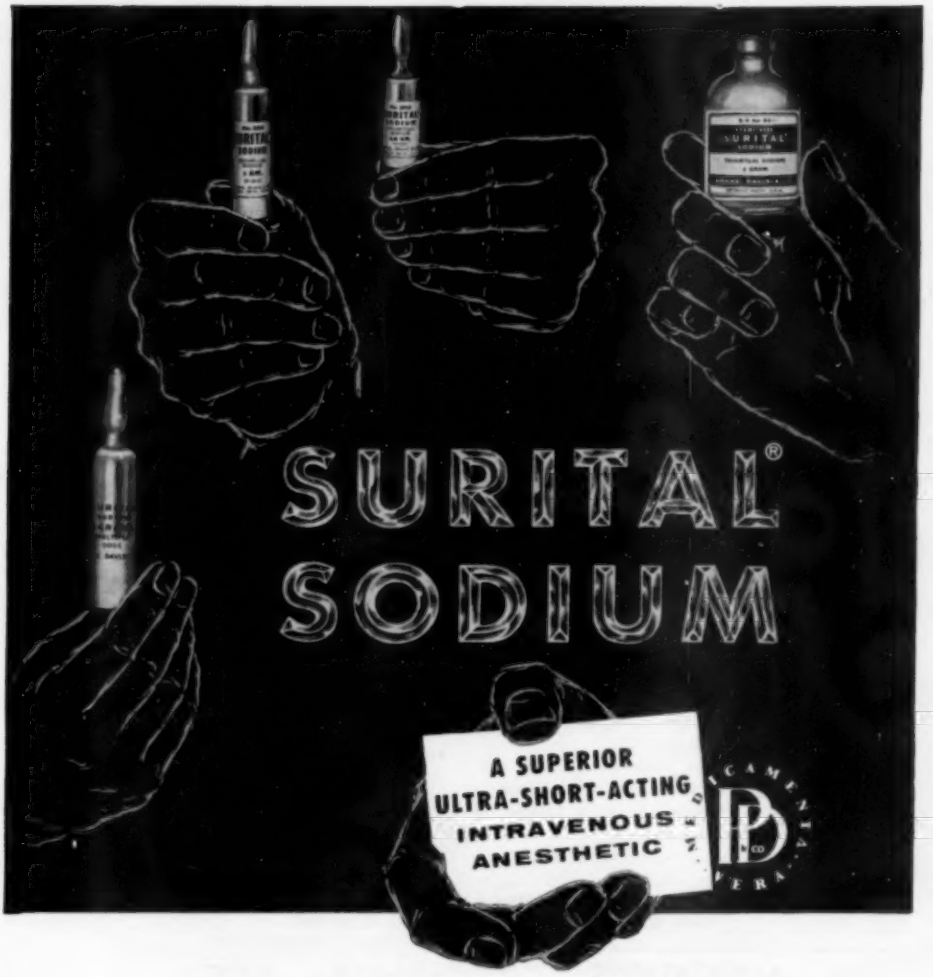
★Coleman P. Calloway (KCV '15), San Antonio, Texas, died on Aug. 18, 1957.

Fred H. Conover (ONT '34), Bloomington, Ill., died Aug. 20, 1957.

Edward W. Elliott (ONT '10), Park River, N. Dak., died in July, 1957.

Lambert T. Rogers (UP '12), Stamford, Conn., died Sept. 3, 1956.

James A. Thom (CVC '05), North Bend, Neb., died on June 14, 1957.



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PROFESSIONAL LITERATURE AVAILABLE ON REQUEST

Department of Veterinary Medicine

PARKE, DAVIS & COMPANY

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STUDENT CHAPTER ACTIVITIES

Student Preconvention Conference Report.—On Aug. 18, 1957, the student and auxiliary delegates met at Ohio State University for a preconvention get-together. All of the schools were represented except Georgia. The married delegates were guests of the married senior students while the Omega Tau Sigma fraternity was host for the single delegates.

A luncheon was served by the student auxiliary in the faculty lounge of the new veterinary laboratory, Sisson Hall. A tour was conducted through the new building and then the group was taken around the campus to see some of the highlights. After the tour, a tea was held by the faculty wives in the student union building.

At a banquet in the evening held at the Desert Inn, Dean Leo L. Rummell, College of Agriculture, was the guest speaker. He showed colored slides and spoke on the new agriculture and veterinary medical program in India.

On Monday evening, August 19, delegates from student chapters and student chapter auxiliaries were guests of the AVMA at a dinner held in the Empire Room, Hotel Cleveland. Faculty representatives and auxiliary sponsors also attended the dinner.

Following the dinner, Mr. D. W. Porter explained the AVMA Group Insurance program and General J. A. McCallam discussed the Veterinary Corps status and matters pertaining to military procurement of veterinarians.

The delegates from the student auxiliaries subsequently met with representatives of the Women's Auxiliary to the AVMA to discuss a number of programs in which they are engaged.

A total of 42 students registered at the Cleveland meeting.

s/Thomas A. Wittich,
Ohio State Student Chapter.

APPLICATIONS

Applicants—Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. This certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative Bylaws.

AMOS, N. S.
Kirkton, Ont.
D.V.M., Ontario Veterinary College, 1949.

BERTAKIS, DEMETRE J.
5408 Telegraph, Oakland, Calif.
D.V.M., University of Parma (Italy), 1944.

BLAU, SHELDON
115 East 12th Ave., Pine Bluff, Ark.
D.V.M., Middlesex University, 1945.

BLUM, SANFORD R.
3219 N. Clark St., Chicago, Ill.
D.V.M., Middlesex University, 1944.

BOURKE, CLARENCE D., JR.
1840 E. Lancaster, Fort Worth, Texas.
D.V.M., Texas A. & M. College, 1952.

CAMERON, WILLIAM B.
3265 Bloor St., W., Toronto, Ont.
D.V.M., Ontario Veterinary College, 1935.

DIAMOND, LEWIS R.
77-46 164 St., Flushing, N.Y.
D.V.M., Alabama Polytechnic Institute, 1940.

FARNHAM, JOHN A.
R.F.D., South Windsor, Conn.
D.V.M., Michigan State College, 1940.

FOLEY, JOHN
1910 Forsythe Ave., Monroe, La.
D.V.M., Oklahoma State University, 1952.

JENNY, JACQUES
Green Valley Farm, Unionville, Pa.
D.V.M., University of Zurich, 1942.

KALTER, HAROLD S.
911 Main St., Holyoke, Mass.
D.V.M., Middlesex University, 1942.

LEOPOLD, SEYMOUR
326 Cross St., Fort Lee, N.J.
D.V.M., Middlesex University, 1944.

LEVINE, LEONARD R.
206 E. 60th St., New York 22, N.Y.
D.V.M., National Veterinary School of Alfort, France, 1951.

LORD, GEOFFREY H.
Johnson & Johnson Research Foundation, New Brunswick, N.J.
D.V.M., Ontario Veterinary College, 1949.

McCASLIN, JOHN H.
R.D. 1, Box 839, West Chicago, Ill.
D.V.M., Middlesex University, 1943.

MICHAELSON, SOLOMON
50 Mandy Lane, Rochester, N.Y.
D.V.M., Middlesex University, 1946.

MOGLIA, RICARDO M.
Feliciano Rodriguez, 2658 piso 1. ap. 3, Montevideo, Uruguay.
D.V.M., Facultad de Veterinaria de Montevideo, 1952.

PALLOTTI, FRANCIS T.
3790 Main St., Hartford, Conn.
D.V.M., Middlesex University, 1944.

PIEKARSKI, JOHN P.
Simmonds Road, Murrayville, B.C.
D.V.M., University of Zurich, 1946.

RUSSO, RAYMOND
192 Main St., Kingston, Mass.
D.V.M., Middlesex University, 1944.

SCHWARTZMAN, IRWIN W.
New Britain Ave., Plainville, Conn.
D.V.M., Alfort Veterinary College, 1951.

SHIRE, JACK
266 Roosevelt Rd., Glen Ellyn, Ill.
D.V.M., Middlesex University, 1944.

SLADE, EDWARD G.
334 Concord St., Framingham, Mass.
D.V.M., Middlesex University, 1943.

STEINBACH, KARL F.
Conyngham, Pa.
V.M.D., University of Pennsylvania, 1939.

TAYLOR, JAMES C.
783 Thornhill Dr., Cleveland, Ohio.
D.V.M., Ohio State University, 1953.

URBONAS, MAX
P. O. Box 56, Sedgewick, Alta.
D.V.M., Hannover Veterinary College, 1947.

VIERA, EMILIO E.
Calle 25, No. 1212 Apto. 3, Vedado, La Habana, Cuba.
D.V.M., University of Cuba, 1952.

WAYNE, IRWIN W.
451 Quincy Ave., E. Braintree, Mass.
D.V.M., Middlesex University, 1944.

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ORGANIZATION SECTION

Applicants — Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and names of his endorsers.

First Listing

MUNOZ, ARMANDO
Casilla No. 3983, Guayaquil, Ecuador
D.V.M., University of Chile, 1941.
Vouchers: Lloyd H. Scrivner, John S. McDonald

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

First Listing

University of Colorado

JONES, WILLIAM E., D.V.M.
Rt. 2, Box 29, Fort Collins, Colo.
Vouchers: Lloyd C. Moss and B. G. Erwin.

University of Pennsylvania

LEMOS, EDWARD J., V.M.D.
Horseneck Rd., Dartmouth, Mass.
Vouchers: Carl E. Chase and R. W. Smith.

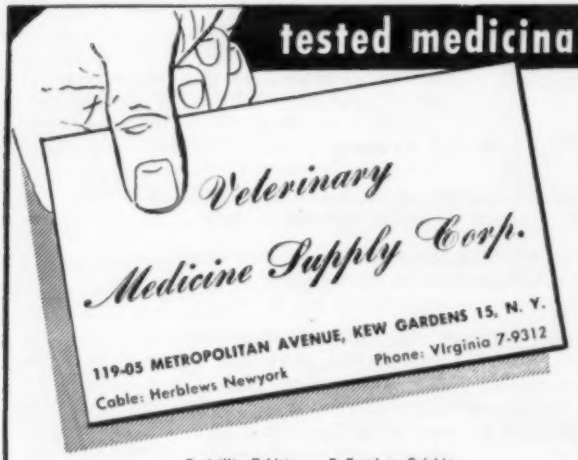
Tuskegee Institute

SMITH, LORRAINE E., D.V.M.
4123 - 4th St., N.W., Washington, D.C.
Vouchers: Edward T. Braye and Rodney T. Gross, Jr.
DAWKINS, WHIT M.
326 W. 19th, South Sioux City, Neb.
Vouchers: John M. Aiken and Paul L. Anderson.

Ciba's Veterinary Products

The veterinary products of Ciba Pharmaceutical Products, Inc., are now marketed (as of July 1, 1957) through ethical veterinary distributors throughout the country. Promotion of these products is carried out by direct mail, journal advertising, and by exhibits at major veterinary meetings.

Ciba now markets 14 products for general veterinary use. A therapeutic index containing detailed information about their veterinary line may be obtained by writing to the company. New veterinary products have been investigated during the past year by the company's veterinary research division under the direction of Dr. Alfred E. Earl (COR '41).



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100 tablets, 500,000 units each (500 tabs. 28.00) (Buffered only) 6.00
100 Penicillin, 100,000 units with Triple Sulfa (500 tabs. 20.00) 4.50
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Combiotic 10cc 5 Dose Vial—4:½ .68c
(10 Vials — 6.30) (100 Vials — 60.00)
5 Gram Dihydrostreptomycin Solution
1 Vial .75c
10 Vials 7.30
100 Vials 70.00
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(Nationally known brand) 100's 22.49
Hydrocortisone Tablets — 20MG
100's 10.50
10CC Vit. B 12 Crystaline 100MCG per CC
Vial 3.05
10 Vials 29.50
50 Vials 139.00
10CC Mercuhydrin Sodium Vial 1.10
10 Vials 10.25
100 Vials 96.00
100 Prednisolone Tabs 5 MG 16.50
1000's 110.00
50 Miltown 400MG 3.05
(10x50 tabs. 29.50) (50x50 tabs. 145.00)
50 Miltown 200MG 2.50
(10x50 tabs. 24.50) (50x50 tabs. 118.00)
50 Equanil 400MG 3.05
(10x50 tabs. 29.50) (50x50 tabs. 145.00)
50 Equanil 200MG 2.50
(10x50 tabs. 24.50) (50x50 tabs. 118.00)

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COMING MEETINGS

Interstate Veterinary Medical Association. Annual meeting. Sheraton-Martin Hotel, Sioux City, Iowa, Nov. 7-8, 1957. K. W. Smith, 510 W. 19th St., Sioux City, secretary.

Animal Care Panel. Annual meeting. Bellevue Hotel, San Francisco, Calif., Nov. 7-9, 1957. R. J. Flynn, Box 299, Lemont, Ill.

Midwest Small Animal Association and the Regional A.A.H.A. Annual meeting. Hotel Burlington, Burlington, Iowa, Nov. 13-14, 1957. J. Porter Coble, 2828 S. MacArthur Blvd., Springfield, Ill., secretary.

United States Livestock Sanitary Association. Sixty-first annual meeting. Chase Park Plaza Hotel, St. Louis, Mo., Nov. 13-15, 1957. R. A. Hendershott, 33 Oak Lane, Trenton, N. J., secretary-treasurer.

Pennsylvania, University of. Fifty-eighth annual conference of veterinarians. Jan. 7-8, 1958, School of Veterinary Medicine, University of Pennsylvania, Philadelphia. Dr. James Mark, chairman.

Cornell University. Annual conference for veterinarians. New York State Veterinary College, Ithaca, Jan. 8-10, 1958. W. A. Hagan, dean.

Ohio State Veterinary Medical Association. Seventy-fourth annual meeting. Netherland Hilton Hotel, Cincinnati, Jan. 8-10, 1958. Harry C. Sharp, executive secretary.

Oklahoma Veterinary Medical Association. Annual meeting. Hotel Lawtonian, Lawton, Jan. 9-10, 1958. Mrs. Larma Bennett, 2805 S. W. 51 St., Oklahoma City, executive secretary.

Kansas Veterinary Medical Association. Annual convention. Hotel Broadview, Wichita, Jan. 12-14, 1958. K. Maynard Curts, 5236 Delmar Ave., Kansas City 3, Kan., secretary.

Tennessee Veterinary Medical Association. Annual meeting. Hotel Andrew Jackson, Nashville, Jan. 12-14, 1958. H. W. Hayes, 5009 Clinton Pike, Nashville, secretary-treasurer.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Des Moines, Jan. 14-16, 1958. F. B. Young, Waukee, Iowa, secretary.

Indiana Veterinary Medical Association. Annual meeting. Hotel Severin, Indianapolis, Ind., Jan. 16-17, 1958. L. M. Borst, 3315 Shelby, Indianapolis, secretary.

Intermountain Veterinary Medical Association. Annual meeting. Hotel Utah, Salt Lake City, Jan. 16-18, 1958. R. A. Bagley, 4600 Creek View Dr., Murray, Utah, secretary.

Minnesota Veterinary Medical Association. Annual meeting. St. Paul Hotel, St. Paul, Jan. 20-22, 1958. P. S. Pomeroy, School of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn., secretary.

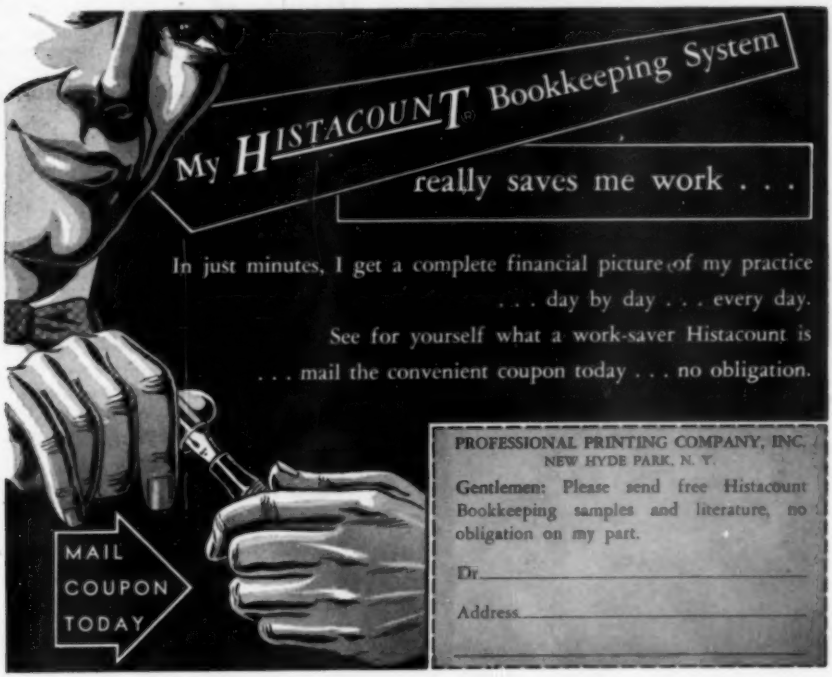
Michigan State University. Thirty-fifth annual postgraduate conference for veterinarians, Jan. 22-23, 1958. College of Veterinary Medicine, Michigan State University, East Lansing. W. W. Armistead, dean.

North Carolina State College. Conference for veterinarians. North Carolina State College, Raleigh, Jan. 28-31, 1958. C. D. Grinnells, chairman.

Oregon Veterinary Medical Association. Winter meeting. Portland, Jan. 31-Feb. 1, 1958. Edward L. Holden, P. O. Box 445, Oswego, secretary.

Wisconsin Veterinary Medical Association. Annual Meeting. Pfister Hotel, Milwaukee, Wis., Feb. 10-12, 1958. Robert Curtis, 419 W. Cook St., Portage, secretary.

Colorado State University. Nineteenth annual veterinary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Trasentine®-Phenobarbital

Vioform® (iodochlorhydroxyquin CIBA)

Entero-Vioform®

C I B A SUMMIT, N.J.

conference. Glover Veterinary Hospital, College of Veterinary Medicine, Colorado State University, Fort Collins, Colo., Feb. 17-19, 1958. Lloyd C. Moss, head, Department of Medicine, Colorado State University, secretary.

Illinois State Veterinary Medical Association. Annual convention. LaSalle Hotel, Chicago, Feb. 17-19, 1958. C. B. Hostetler, 1385 Whitcomb Ave., Des Plaines, Ill., executive-secretary.

American Animal Hospital Association. Silver anniversary meeting. Drake Hotel, Chicago, Ill., April 23-26, 1958. Joseph A. S. Miller, president.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2. Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December at 3004 16th St., San Francisco, Calif. Mr. Herb Warren, executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 99 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. C. Lawson, 4040 El Camino, Palo Alto, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

Russian Dogs Travel in Rockets

Journalists who attended a Moscow press conference in June, 1957, in connection with the International Geophysical Year, were shown these dogs said to have travelled in rockets which reached a height of over 62 miles.

Schering Corporation and White Laboratories Merge

Stockholders of Schering Corporation and White Laboratories, Inc., voted in separate meetings on Sept. 19, 1957, to merge the two firms into a single pharmaceutical company with current consolidated annual sales in excess of $75,000,000. The merger became effective immediately, it was announced jointly by Francis C. Brown, president of Schering, and J. C. DeCesare, president of White Laboratories.

According to the joint announcement by Mr. Brown and Mr. DeCesare, White Laboratories and its subsidiary, Pharmaco, Inc., will continue to operate under their present names. Activities of Schering's proprietary drug subsidiary, Union Pharmaceutical Co., will be merged with those of Pharmaco. Thus, Schering now has three organizations serving a broadened market through research, manufacture, and sale of ethical and proprietary drugs, the joint statement said.

Two-way radios are being used by mounted livestock buyers at the Chicago stockyards, so they can talk to each other as they ride around the yard pens. The battery operated unit (8 lb.) is encased in a special saddle bag with both earphones and a loud-speaker and a range of up to 2 miles.—*Am. Cattle Producer (Sept., 1957): 19.*

Chas. Pfizer & Co., Inc., Contributes $16,000 to AVMA Research Fellowship

The Board of Trustees of the AVMA Research Fund announced during the Cleveland convention that a grant of $16,000 from Chas. Pfizer & Co. Inc., made in 1955, had been accepted following an understanding as to the utilization of the grant for fellowship purposes.

The grant will be used to activate one or two (maximum) fellowships per year in areas of research activity mutually agreeable to the trustees, the Fellowship Committee, and the donor. The grant has been designated as the "Chas. Pfizer Gail Hawley Memorial Research Grant" in memory of the late Dr. Gail E. Hawley, veterinary consultant at the company's agricultural research center at Terre Haute, Ind., who died early in 1957 from injuries suffered in the crash of a chartered plane.

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Supplied: 30-cc. multidose vials, each cc. containing 50 mg. of Gallogen.*

Dosage: Intramuscular or intravenous administration. Cattle—500 mg. (10 cc.)
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(*Diethanolamine salt of paratolylmethylcarbinol camphoric acid ester.)

VETERINARY DIVISION
THE S. E. MASSENGILL COMPANY
BRISTOL, TENNESSEE

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, in St. Petersburg. R. L. Brutus, 336 E. 15th St., Hialeah, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Barrow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

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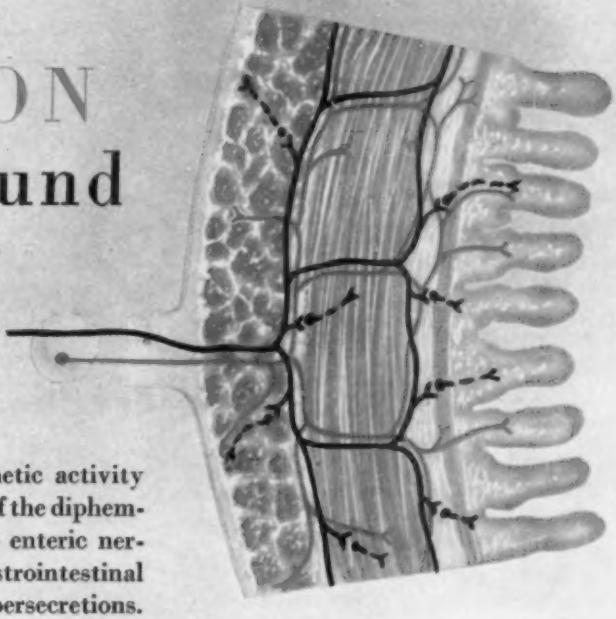

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Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

East Central Iowa Veterinary Medical Society, the second Tuesday of every month. Dr. W. T. Rugger, Oxford, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Winneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

KANSAS—Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

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Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

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MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5834 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Chester R. Pleggs, 4249 Peck St., St. Louis 7, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

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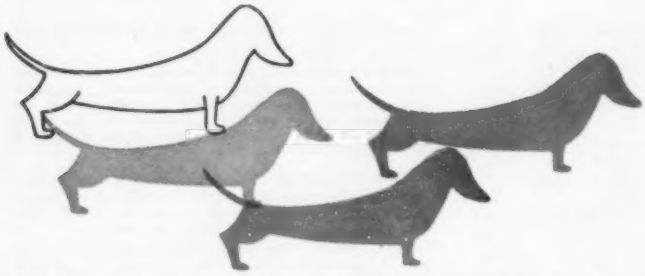

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1. Rachman, M., and Frucht, T. R.: Vet. Med. *49*:341, 1954.

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Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College. Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Thursday of each month in the Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. James R. Burgess, Rockingham, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

Stark County Veterinary Association, the second Monday of each month. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forrest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Willamette Veterinarian Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Dr. Marvin Corff, McMinnville, secretary.

PENNSYLVANIA—Del-High Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August, O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). R. J. O'Hern, P.O. Box 617, Cumberland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April, William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

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REFERENCES: 1. Mosier, J. E.: Vet. M. 50:605 (Nov.) 1955.
2. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956.
3. Pollock, S.: J. Am. Vet. M. Ass. 129:274 (Sept.) 1956.

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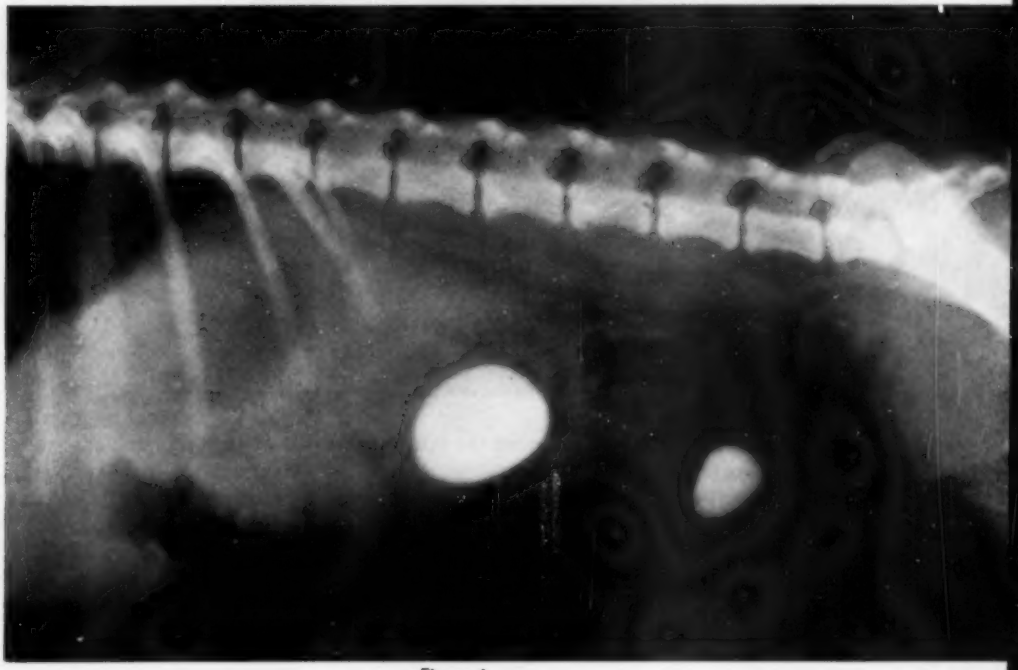

Figure 1

History.—A nonspayed, female Wire-Haired Fox Terrier, 5 years old, had access to a graveled yard. The dog refused to eat, exhibited abdominal pain, and vomited three times in ten hours. Two hard objects could be palpated in the abdominal cavity, presumably in the small intestine. A week previously, the dog had a bowel obstruction from a stone it had swallowed. The stone passed unaided 24 hours later. This radiograph was taken in an effort to determine whether surgery would be necessary to relieve the obstruction.

(Diagnosis and findings are reported on next page)

Here Is the Diagnosis

(*Continued from preceding page*)

Diagnosis.—Bowel obstruction. The objects, because of their density and the evenness of their contours, were thought to be stones.

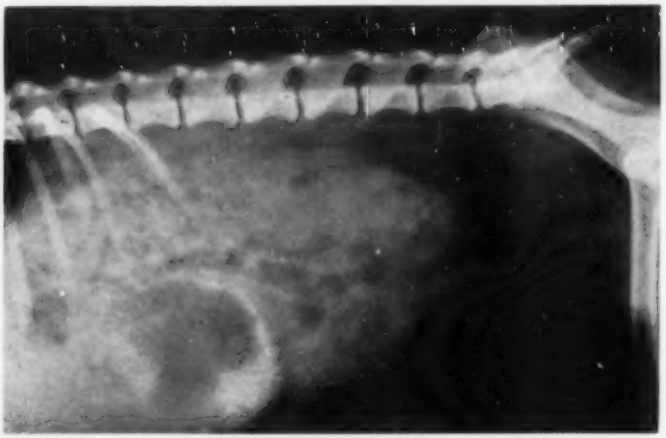

Fig. 2—Radiograph (lateral view) of the abdominal region of the dog taken after the stones had been expelled.

Comment.—It was believed that the larger stone was of such proportion that it could not pass through the intestinal tract, so surgery was advised.

The owner elected to delay surgery since the previous stone, although smaller, had passed satisfactorily. No treatment was given and, surprisingly, on the following morning both stones were passed and found in the cage; the dog was ready to eat. A radiograph (fig. 2) taken then confirmed that the stones had passed.

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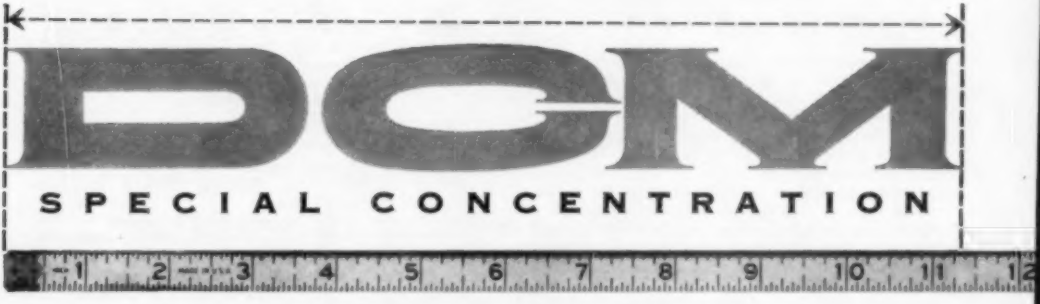

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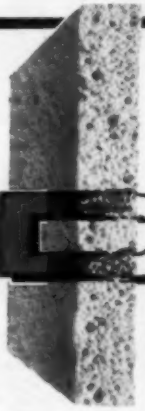

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Safe • Effective

- **Adequately controls bleeding during extraction of teeth, or for tartar removal.**
- **Eliminates need for constant blotting and wiping of bleeding gums.**
- **Keeps field dry, providing clear visibility for easier completion of dental manipulation.**
- **Easy to use . . . Safe, fast and effective . . . Economical too!**

AVAILABILITY: *Solusponge Dental Packs* • *Solusponge Dental Granules* • *Solusponge Dental Powder*

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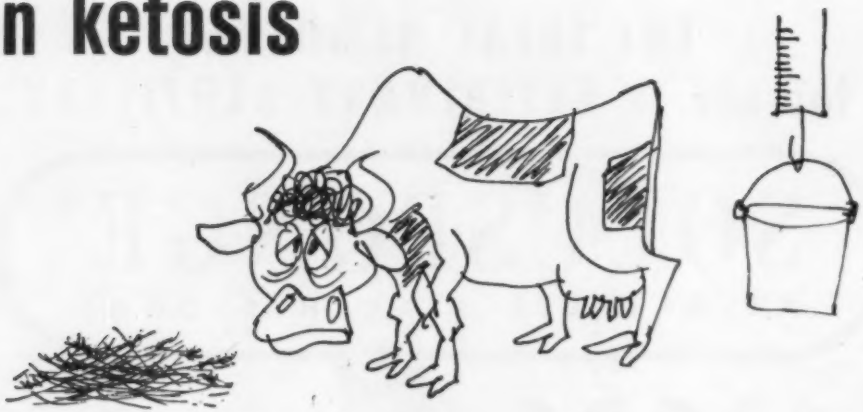
Parlam CORPORATION • 340 Canal Street, New York 13, N. Y.

SOLE CANADIAN DISTRIBUTOR: Veterinary Medical Supply Co. of Canada, 6579 Somerled Ave., Montreal, Que.

in ketosis



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STERANE®

BRAND OF PREDNISOLONE

to put her back on feed and restore milk volume faster

STERANE—for an early return to milk production.

STERANE, three to five times more potent than hydrocortisone and cortisone, stimulates glucose formation and helps re-establish physiological equilibrium. In clinical studies a single intramuscular injection of *50* to *100* mg. of Sterane promptly restored energy metabolism, with rapid reversal of ketotic symptoms in over 83 per cent of 299 cases treated.

STERANE INTRAMUSCULAR-VETERINARY *Sold to veterinarians only*

Pfizer

Department of Veterinary Medicine, PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc.*, Brooklyn 6, N. Y.

Well-established small animal hospital and home in New York State for sale. Excellent large animal potential. $38,000. Write for particulars. Address "Box S 2," c/o JOURNAL of the AVMA.

Excellent two or three man practice for sale, with a completely equipped large and small animal hospital in central Oklahoma. $60,000. Address "Box S 5," c/o JOURNAL of the AVMA.

Established practice for sale in Minnesota. Gross average $20,000. Office in home. Priced at value of real estate. Equipment and inventory optional. Address "Box S 16," c/o JOURNAL of the AVMA.

Busy mixed practice for sale, 90% large animal, situated in small, friendly town 20 miles northeast of Guelph, Ont. Full price $20,000, includes home, office, surgery, dispensary well-stocked with drugs, and equipment. Address "Box S 17," c/o JOURNAL of the AVMA.

To insure prompt delivery, replies should be carefully addressed: Complete box number as given in the ad, AVMA, 600 S. Michigan Blvd., Chicago 5, Ill.

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Gummed labels on roll printed to order; handy dispenser. Quantity buying cheaper—store safely through polyethylene protection. Money back guarantee. Postcard brings samples. Boyd's Printery, Box 462B, Genoa, Ohio.

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Professional assistant and veterinary correspondent —Promising opportunity offering permanent employment to person with pre-veterinary or biological science degree. Related commercial experience desired. This position involves assisting the professional staff with administrative duties and handling selected division correspondence on veterinary specialty products. If interested, please send resume at once to: Personnel Director, Eaton Laboratories Division, The Norwich Pharmacal Company, Norwich, New York.

Too Late to Classify

Assistant wanted for busy small animal practice in southern California. Modern, well-equipped hospital. Good salary and excellent opportunity for future advancement. Address "Box S 15," c/o JOURNAL of the AVMA.

Veterinarian wanted for staff of large hospital for small animals in New York City. New York license required. Opportunity. Address "Box S 18," c/o JOURNAL of the AVMA.

Dr. Sinha Joins Jensen-Salsbery Laboratories

Dr. S. K. Sinha, a graduate in veterinary medicine from the University of the Philippines, has joined the staff of the Jensen-Salsbery Laboratories, Kansas City, Mo., as research director. He obtained his master's and doctor's degrees in virology at the Department of Veterinary Science, University of Wisconsin.

Dr. S. K. Sinha

Prior to his association with Jen-Sal, Dr. Sinha directed the ARS Regional Diagnostic Laboratory at Philadelphia, then served an assistant professorship at the University of Pennsylvania. Later, he was associated with tissue culture research on coryza viruses in man.

Dr. Sinha, his wife, and two children have established residence in Shawnee, Kansas.

Standards for Production of Laboratory Mice.—Increased anti-cancer and other research has created a demand for inbred mice which exceeds the production capacity of special genetic laboratories. It has, therefore, become necessary to devise methods by which commercial laboratory mouse producers could cooperate in producing inbred mice. To facilitate this endeavor, a committee has compiled an outline "Minimum Standards for the Commercial Production of Randombred and Inbred Laboratory Mice" which can be obtained from: Executive Secretary, Institute of Laboratory Animal Resources, National Academy of Sciences, 2101 Constitution Ave., N.W., Washington 25, D. C.

r

rabies

R_L Rabies Vaccine

Modified Live Virus—Chick Embryo Origin

Made with a true modified rabies virus cultivated in chick embryos, this vaccine is absolutely incapable of producing disease. At the same time, it produces high-degree immunity which has lasted in field tests beyond 2 years!

With the exclusive RECONSTIQUICK FEATURE for convenience! These vaccines reconstitute immediately upon introduction of the diluent, going into a fine homogenous suspension which will pass easily through a 24 gauge needle.

Vacuum Dried . . . for safety!

Phenolized—20% Tissue Suspension

A finely ground homogenous suspension of brain and spinal chord from sheep and goats previously inoculated with Fixed Rabies Virus, National Institute of Health Strain. The virus in this vaccine is chemically inactivated with Phenol. For administration in routine yearly vaccination of pets and/or after exposure to the disease.

All R_L Products offer safety, efficiency and reliability in the prevention and treatment of animal disease!

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Research Laboratories, Inc.

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Quick relief for Bone, Bursal or Tendon Lameness

Single Bottle.....$2.00
3 and 1 free.....5.00
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12 and 4 free.....17.00
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Hudson, N. Y.

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Features precision-made hydraulic unit guaranteed for life of the table. Finger-touch control raises, lowers and locks table in seconds. Large 24" x 60" top. Movable on concealed casters.

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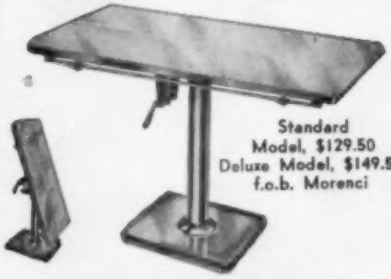

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Chrome plated single pedestal and base combined with reinforced stainless steel top. Positive locking adjustment in twelve positions. Super size 24" x 60" top.

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new cell propagation technique CELLORIGEN* marks significant milestone in advancement of basic immunology

Development of Cytohep (Jen-Sal) marks the initial use of modern tissue culture methods in the production of a canine hepatitis vaccine. Constant control inherent in Jen-Sal's exclusive CELLORIGEN process guarantees a protective agent which is strongly immunogenic, safe and pure.

Controlled cell propagation greatly enhances antigenic properties while retaining the confirmed safety of a killed virus. The possibilities of reversion to virulence, virus shedding in vaccinated animals, and of vaccine contamination from extraneous virus are eliminated.

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A single 2 cc. subcutaneous or intramuscular injection two weeks after weaning builds a high level of protection against hepatitis during the critical period of a dog's life.

Order Cytohep in cartons of six single dose vials from any Jen-Sal Branch or representative.

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(tissue culture origin)

Jen-Sal

* superior antigens of cellular origin

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